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In Defense of the Stethoscope*

By James B. Herrick, *Chicago*

THERE is a tendency nowadays to attack the stethoscope. It is criticized in print, in addresses in medical societies and in conversation between doctors. As a result it is losing caste as an instrument of diagnosis. Moreover, its loss of prestige carries with it a lessened appreciation of physical diagnosis in general, once a most highly regarded feature in medicine.

The charges against the stethoscope are two: that it is antiquated, its place taken by other methods especially by the x-ray, and that it does more harm than good because its findings are so often misinterpreted.

As to the first charge that it has been, and should be, displaced by the x-ray and other methods of diagnosis:—

For diagnosis we must rely as heretofore upon the history and physical examination, with free recourse to instruments of precision, the laboratory, the therapeutic test, or even the exploratory surgical procedure. In this complex of diagnostic agencies the stethoscope and physical diagnosis have a part that is decidedly non-negligible, though we must recognize the fact that the relative values of these different methods have undergone many changes. Thus in tuberculosis the stress formerly laid on personal and family history and physical examination has been largely transferred to the examination of sputum and the study of x-ray films. The Wassermann test and x-ray have assumed prominent rôles in the recognition of thoracic aneurysm. The x-ray may show a tuberculous focus or an aneurysm that has been missed by the other methods, or it may be decisive in determining the extent of such lesions.

There is however, another side to this question. The stethoscope may discover disease that escapes the x-ray. A few râles at an apex, associated with a cough, slight temperature and expectoration may be more convincing than the film that shows no lesion or only suggests it. A pleural or pericardial friction or a diastolic murmur may furnish unmistakable evidence of organic disease that is not shown by x-ray. No x-ray film or even fluoroscopic examination can convey the sense of strength or weakness that is revealed when the trained hand palpates the region of the laboring heart or the stethoscope detects the murmur at apex or base and brings out the character of the closing sounds of aortic and pulmonic

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valves. The x-ray discloses the size and shape of the heart, but not its behavior in action, at least as regards sound and strength. To see a photograph of a tired horse stumblingly hauling a heavy load up a hill gives evidence as to the efficiency of the horse. In addition, to hear him puff and wheeze, to hear the wagon creak and the driver's whip crack yields important information as to the ability of the horse to carry the load to its destination.

The electrocardiograph is of great value, to be used more and more until perhaps employed in every complete examination as much as pulse counting, blood pressure reading, urinalysis. Yet many facts concerning the heart and its efficiency are not recorded by the electrocardiograph. They are discovered only by physical examination and a study of the history, which methods must still be regarded as our most useful means of diagnosis of cardiac disease.

No defender of the stethoscope should take the false position that this instrument is arrayed *against* the x-ray or electrocardiograph. The correct attitude is to admit the superiority of these other instruments in many respects, to welcome their help. The stethoscope, the x-ray and electrocardiograph should be allies and not enemies.

That other instruments have shown the fallibility of the stethoscope is cause not for discouragement but for stimulation. Efforts should be made to improve technique and interpretation of findings. Formerly such interpretation was possible chiefly through post mortem examination. Now it can be made earlier by instrumental and laboratory aid. We should not allow the stethoscope and all it stands for to go into the discard because forsooth much of the information may be gained by other lazier and supposedly more exact methods. We should not teach our students, undergraduate and graduate, that the first thing to do in diagnosis is to rush to the x-ray and electrocardiograph any more than we should teach them that the first thing to do in an abdominal pain is to explore the abdomen surgically. This is shirking duty and responsibility and at times leads to grievous error.

We should remember, too, that the interpretation of an electrocardiogram, an x-ray film, a laboratory color reaction, a bit of tissue under the microscope—all these interpretations depend on the action of a human brain. That brain may be in error in the laboratory as well as when connected with the earpieces of a stethoscope. The optic nerve may err as well as the auditory. Also, it is to be noted that properly to put together the results of tests made by different men and different instruments of varying reliability is not an easy task. Strange and unworkable assembled diagnoses sometimes result. For truth has a tricky way of slipping out of hand or getting distorted as it is passed from one individual to another. The more remote from the physician the instrument or laboratory, the greater the danger of error. The stethoscope will be one means of keeping him close to the patient and thus closer to the truth. Furthermore, it

helps him to retain that important something called the personal touch.

The second objection to the stethoscope is that because its findings are so often misinterpreted it does more harm than good.

Again we must admit justice in the criticism. A few râles with a roughened expiratory tone, or a murmur at the apex of the heart, and the uninformed or impulsive doctor, or the over-conscientious doctor who feels that in the interest of his patient he must recognize heart disease or tuberculosis in the incipient or hopeful stage, makes a diagnosis of tuberculosis or heart disease with all the penalties thereunto appertaining:—psychic upset, altered modes of living and change in occupation. On the basis of the one finding, with disregard of other features, he has jumped to a disastrous conclusion that may be wrong. No wonder the critics cry, "Away with the stethoscope."

Now the same argument may be used against almost any other aid to diagnosis, the clinical thermometer, the sphygmomanometer, the Wassermann test, the reaction for albumin in the urine, the leucocyte count, the facts in a history. Are not their results misinterpreted again and again? Some physicians have said the Wassermann reaction was a curse, for at times positive when there was no syphilis, oftener negative when syphilis exists, it has led to many errors. Others have rebelled against the emphasis laid on casts and albumin in the urine. That blood pressure—too high or too low—is made too much of by the laity and many doctors is well known. And the sins committed in getting histories, not alone sins of omission but of commission as well, the wrong interpretation of headache, vomiting, dyspnea, cough! One has no difficulty in making a long list of misinterpretations along these various lines. Why, then, should the stethoscope be the instrument singled out as the criminal whose sentence is to be banishment when others are also guilty, at times even more guilty?

The greatest outcry against the stethoscope has come from those who deplore the fact that the murmur revealed by this instrument so often means nothing pathologic yet is regarded by the injudicious examiner as an indication of disease. I will not labor the trite statements about the so-called accidental murmur and the theories as to its origin. If there is no history of rheumatism, if the heart is not large, if the second tones are normal, if there are no symptoms of cardiac incompetence such as dyspnea, cyanosis or râles, and if the murmur is systolic, we are told to let it pass as not meaning organic heart disease. But suppose it is due to anemia, or fever, or cardiac irritability as in exophthalmic goiter, should it not be noted as a symptom of such condition? Should we in cowardly fashion decline to listen for it or to admit its presence or discuss its significance for fear we may misinterpret it? It is up to us to decide whether it means nothing or something. The fault is in us and not in the stethoscope. We should discard our error and not the instrument.

There is another side to the question of the systolic heart murmur. Let us admit that its importance as

an indication of heart disease has been, and still is in many quarters, overstressed. Yet who among us does not in practice value highly the information afforded by murmurs, including those heard in systole? All agree that diastolic murmurs are of great significance as indicating organic disease. We must also insist that many systolic murmurs command our respectful attention because they mean something.

I am inclined to think there is developing a tendency to underestimate the importance of the systolic murmur as an evidence of heart disease. Other signs, such as increase in size of the heart, altered second tones may be trivial or absent. The heart may for the time be competent so that there is no dyspnea. Yet organic disease, such as syphilitic or rheumatic, may lurk in the valves or myocardium. The murmur rightly interpreted may lead to more thorough investigation of the case. A word of caution—not a note of alarm—may be given and the patient saved from a later breakdown. Not a few young men were admitted to active service in the Great War, in spite of a known systolic murmur which was regarded as meaningless. Some of these young men came out of the army fatally wrecked because of cardiac breakdown.

One other point. Granting that the murmur is by no means the sole criterion of a disease of the heart or of a heart's efficiency, it is worth something in a negative way to examine and find no murmur. It may be a comforting bit of evidence that the heart is all right. We do not exclude tuberculosis because the thermometer, the sputum examination or the x-ray fail to show change from the normal, but we rightly give these negative findings weight in trying to reach a decision. Similarly the stethoscope has a function to perform in excluding the presence of murmurs.

To conclude, this is a plea for the sane use of every means that may help in diagnosis, including the stethoscope and all that it stands for in the way of physical diagnosis. Physical examination should not yet be regarded as displaced by other methods. It still has a legitimate function. Undergraduates and practitioners should still be taught its theory and its practice so that it may not become a lost art.

The Use of Iodine in Exophthalmic Goiter*

By J. H. Means, M.D., F.A.C.P., *Boston*

As a result of studies on the action of iodine in exophthalmic goiter begun in the spring of 1923, when Plummer first described its characteristic effect, and continued ever since, we have come to certain conclusions about indications for treatment which seem sufficiently definite to warrant review. What iodine does has become clear; how it does it remains a mystery.

The most characteristic phenomena which this element or its salts produces in persons suffering from real Graves' disease are the rapid amelioration of symptoms, especially the nervous, and the fall in pulse rate and basal metabolic rate which occurs, with but few exceptions, when adequate dosage is employed in patients who are not already under its influence. We have come to look upon this response as one of the earmarks of the disease, and when it fails to occur, at least in some measure, we doubt whether we are dealing with thyrotoxicosis of the true exophthalmic goiter type. It is a therapeutic response as rapid and as specific as that of myxedema to thyroid, pernicious anemia to liver extract, or scurvy to orange juice.

The rate at which and the extent to which the intoxication is ameliorated depends in part at least upon the intensity of the latter before iodine is given, and also upon the geographic area in which the case originates. The nervous symptoms may show improvement within a matter of hours, and the metabolism, as we find it in Boston, a non-goitrous region, drops about five points a day in the severely toxic and about three points a day in the moderately toxic patients, the full effect being reached in from eight to ten days[1]. In other words, the farther the state of the subject is removed from normal, the greater is the effect of the corrective agent. This principle applies to many things in medicine. An agent may have little or no effect in the normal and a great one in the diseased. That the characteristic iodine response may be regarded as specific in exophthalmic goiter is indicated by the fact that iodine has no effect on hyperthyroidism induced in man or animals by thyroid feeding[2, 3, 4], and an uncertain or absent one in what may be taken to be pure adenomatous goiter with hyperfunction.[5]

The matter of locality in relation to the iodine response is of great importance. The severity of the disease

*From the Thyroid Clinic of the Massachusetts General Hospital.

†Read at the Minneapolis Meeting of the American College of Physicians, February 12, 1930.

is unquestionably greater in goitrous than in non-goitrous regions[6]. This is shown by the higher operative death rate in the days before iodine preparation, the greater frequency of toxic crises, and, as recently shown by Coller[5], the less striking response to iodine in cases arising in goitrous areas. When one uses iodine in exophthalmic goiter these geographic differences must always be borne in mind. They are, however, differences in intensity or degree, I believe, rather than in kind; the same relations hold in regard to the action of the drug in the goitrous as in the non-goitrous portions of the globe, but in the former the extent and rapidity of its action are likely to be less.

Certain other facts about the characteristic iodine response deserve emphasis. For one thing, it is important to recognize that the response may be greater in a given individual at one stage of his disease than at another, also that it may occur in quite characteristic form late in the course of the disease as well as near its beginning. It is my conception of the action of iodine in exophthalmic goiter that at any given time during the course of the disease it reduces to a certain extent the intensity of the thyrotoxicosis, and that at any other time it may reduce it to the same or to a greater or a less extent. I further believe that it has no effect whatever on the rate of progress of the disease. Iodine does not tend to shorten it. It diminishes the fierceness of the flame but in no way affects the duration of its burning[1]. Patients may have increasing thyrotoxicosis while taking iodine, but I believe that that is in spite of, not because of the drug. I have never seen patients with exophthalmic goiter made worse by iodine, nor have I ever been completely convinced that they truly become intolerant or refractory to it. The action of iodine in exophthalmic goiter bears some crude analogy to that of digitalis in heart disease or salicylates in rheumatic infection. These drugs diminish the intensity of symptoms but do not alter the progress of the diseases in which they are used.

The dosage of iodine necessary to bring about the characteristic response is a matter of some interest. It, like the severity of the disease, very likely varies from place to place, being greater perhaps in goitrous than in non-goitrous regions. The matter has been studied in our Thyroid Clinic[7] and it was found that in twelve out of thirteen patients with exophthalmic goiter as great a reduction in basal metabolism was obtained with one drop of Lugol's solution daily as with much larger doses. In eight out of twelve other patients, half a drop of Lugol's solution daily was as effective as larger doses. Even doses smaller than half a drop sometimes produced perfectly satisfactory responses.

It seems likely, therefore, that in Boston one drop of Lugol's per day is often an adequate dose. Smaller doses may be effective, but are less dependable. Larger ones accomplish no apparent useful purpose, but at the same time it should be admitted that they do not appear to be harmful. In goitrous districts no doubt the minimum adequate dose is larger; also in our own locality, in persons

in acute thyrotoxic crises, it may be larger. We do not know about this having never dared to give the smaller doses to patients of this type. I mention this matter of the minimum effective dose as a point of scientific interest. I do not wish to be understood as recommending such a dose in the routine treatment of the disease. In contrast to digitalis and salicylate, the difference between the dose of iodine that will have full pharmacologic effect in exophthalmic goiter and the toxic dose is very great. There is hardly any danger of giving too much; therefore, we must be sure not to err on the side of not giving enough. We commonly use doses of five, ten or even fifteen minims three times a day, and in a very toxic case we might give more. The much larger doses sometimes used by Plummer[6] we have not found necessary, but this may be because the disease is commonly more severe in his locality than in ours.

Another question of both theoretical and practical interest is that of whether the chemical state of the iodine administered, or the route by which it enters the body, in any way modifies its effect. From such observations as we have made, the answer would seem negative in each case. Potassium iodide solution containing no free iodine acts just like Lugol's solution; ethyl iodide administered via the lungs produces the same result as Lugol's solution or potassium iodide by mouth.[8]

In view of the various considerations based upon the experience we have had, it seems to us working in the Thyroid Clinic of the Massachusetts General Hospital, that the uses of iodine in exophthalmic goiter may be defined fairly categorically. They seem to us threefold:

First and most important, to prepare patients for operation,

Second, to control residual symptoms after operation, and

Third, as a treatment per se of the disease, in certain selected cases.

Of the first use I need say but little; it is familiar to the profession. One takes it for granted nowadays that the way to treat exophthalmic goiter is to get the full effect of iodine and then do an extensive surgical resection of the thyroid gland at once, continuing the iodine meanwhile. Under such management the risk is slight and the postoperative storm is rare. A single warning, however, may not be amiss. In the relatively few cases where the thyrotoxic manifestations show no sign of diminishing under iodine, and especially in those in which they actually increase, operation should be postponed; patients in this phase may die postoperatively, iodine or no.

The second use, that is to control the postoperative residual thyrotoxic manifestations, is of considerable interest and should be known to the physician. Many patients, either directly or a few weeks after subtotal thyroidectomy, and still more often after less extensive resection, show definite symptoms and signs of residual thyrotoxicosis, perhaps with an elevated, but often with a normal metabolic rate. Nearly always these will disappear on small doses of iodine (Lugol's solution one to three minims per day), and with their disappear-

ance pulse and metabolism drop, the latter perhaps to a definitely substandard rate.[9, 10] Symptoms, pulse, metabolism, and weight have been found repeatedly remarkably sensitive to iodine in these postoperative days, and can be increased or decreased at will, by giving or withholding the drug. Our practice is to give enough to control the symptoms regardless of the metabolic rate. This should be continued until the symptoms fail to return upon the omission of the drug, with us this is generally a matter of not more than a few months. Sometimes the subjective symptoms can be helped by a dose of iodine so small that there is no effect upon metabolism or weight. Patients often can control the dose themselves. Only today I saw a postoperative patient who did not want to take three drops because, although it stopped her symptoms, it made her gain weight, which she did not want to do. It is quite possible that one drop will accomplish the former and avoid the latter effect.

In a few of our cases, and in relatively more in goitrous districts, the postoperative residual symptoms do not yield to iodine and gradually increase. These patients in time usually show a regrowth of thyroid tissue and require further operation. It is the existence of this type, particularly in goitrous regions, that gives the operation of total thyroidectomy[11] some justification, in theory at least.

The third use of iodine, namely as a treatment per se, requires elucidation. There are undoubtedly patients, the course of whose exophthalmic goiter is destined to be so mild that they might go through the course of their disease without inconvenience or ill effect with iodine alone for treatment. If we could recognize these we might so treat them, but we cannot. The fact that exophthalmic goiter starts with a mild course is no guarantee that it will so continue, and in practically every instance where, because of the apparent mildness of the disease, we have carried on with iodine alone, we have later regretted having done so.

There is, however, one small group of patients that may be legitimately treated by iodine alone.[1, 12] These are the very chronic, mild and stationary types. We know that early in its course exophthalmic goiter tends naturally to remissions and relapses, and that later, even without treatment, it tends to burn itself out. There are, however, a few cases in which the fire smolders on for years, true chronic exophthalmic goiter without signs of either getting worse or clearing up. We have seen a few such cases where iodine relieved all symptoms, and in which they could be held in abeyance as long as the patient continued to take the drug. In such cases, which are few and far between, and in no others, do I think that it is permissible to use iodine as a sole treatment for the disease.

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The Association of Chronic Ulcerative Colitis and Multiple Polyps*

By J. Arnold Bargen, M.D., and Mandred W. Comfort, M.D.,
Division of Medicine, The Mayo Clinic, Rochester, Minnesota

IT has been shown that in 10 per cent of cases of chronic ulcerative colitis multiple polyps of the large intestine develop eventually. This estimate is based on a statistical study of 693 cases of chronic ulcerative colitis reported before this association a year ago. The patients in this series had had symptoms of the colitis for months or years and many had not had specific treatment. Just how this percentage will be altered in the future can not be foreseen, but statistics indicate that it will be reduced favorably. It is difficult also to determine what share of total so-called multiple polyposis of the large intestine or "polyposis intestinalis" this represents. An effort to evaluate this and to study factors in the origin of multiple polyps of the large intestine prompted this investigation.

Literature on the subject of polyposis of the large intestine is confusing. Classifications, such as that of Erdmann and Morris, into the adult type (acquired) and the adolescent type (congenital and disseminated), while serving to distinguish the two generally recognized groups, seems to fall short in certain significant features. This classification fills the conventional desire of placing all polyps in main groups, but it fails to take cognizance of origin, manner of growth, or ultimate outcome of any given polyp. This, it seems, should be the aim in classifying a disease as grave as the one in question. The polyps are all of one type, resulting from hyperplasia or hypertrophy of regions of intestinal mucous membrane. If classified from the standpoint of origin, they may be divided into three types: (1) those traceable to definite preëxisting inflammatory disease, such as chronic ulcerative colitis; (2) those arising apparently as innumerable polypoid projections without previous demonstrable inflammation or other disease; this group should include the adolescent and familial types, and (3) those including a few disseminated, adenomatous polyps discovered either at necropsy, or in the investigation of an occasional slight hemorrhage from the rectum or a malignant neoplasm. The first type only will be considered here. Further investigation may place in-

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flammation as the outstanding etiologic factor in the origin of all types of polyps.

Murphy stated: "The etiology of intestinal polypi, like that of the common wart, is shrouded in mystery: Whence they come and why they go is like the riddle of the sphinx." Many factors, however, point to the significance of inflammatory disease or irritation of the mucosa playing a part in the etiology. The hypothesis that there is inherent weakness in cellular growth, and physiologic or chemical extraneous effects,[4] seems plausible but has no practical application. It is Graham's belief that irritation or infection is the basis of the growths and that those of adolescence are due to unusually sensitive mucous membranes. After a careful study of the subject, Schmieden and Westhues concluded that inflammation is the chief, but not the whole, cause. They did not agree with many authors that the Cohnheim theory of embryonal rests applies here but rather that the problem is wholly one of growth, or "misgrowth." Dukes stressed the significance of irritation, and called attention to the close similarity between intestinal polyps and the disseminated regions of hyperplasia on the skin of mice in so-called tar cancers. Erdmann and Morris also stressed the probability that all adult types of polyps could be traced to some type of irritation.

If irritation and infection are factors in the original inception of intestinal polyps, and since in 10 per cent of the cases of chronic ulcerative colitis polyps occur eventually, it would seem that valuable information might be gleaned from a study of these cases.

Justi described "colitis hyerplastica polyposa dysenterica" in which he noted in the lining of the large intestine "plateau-like excrescences and elongated finger-like things," suggestive of polyps. He believed the condition to be the result of bacillary dysentery. His descriptions of two fatal, fulminating cases of chronic ulcerative colitis are excellent. Levin reported three cases of extensive polyposis of the colon in association with chronic ulcerative colitis and emphasized the common occurrence of these two conditions in the same patient. Cripps described a form of polyposis in which the mucous membrane has the appearance "of having been slashed into ribbons left attached at one end." He did not, nor did many of the older writers, distinguish this from the other forms of polyposis. Warwick, in considering etiology, called attention to the fact that the older observers suggested that polyps might be the result of areas of hypertrophied mucosa caught in newly formed cicatricial tissue in healing ulcers.

Saint expressed the opinion that the polyps associated with chronic ulcerative colitis are not true tumors but strips of almost detached mucous membrane and much inflammatory tissue.

Struthers, however, tried to show that chronic ulcerative colitis was one of the chief causative agents in the origin of polyps. He followed the sequence of events thus: There is first severe colitis with undermining ulcers; such ulcers may coalesce and increase in size but portions of mucosa pre-

serve their blood supply. These stud the surface of the large intestine; they heal and become round; fibroblasts are proliferated and begin to contract with resultant cicatrization, then tubules of polypoid projections are occluded, and finally Virchow's "colitis polyposis cystica" results. Elevation of these thickened and altered areas of hypertrophied mucosa results in increased friction and traction which in turn stretches the surrounding adjacent mucosa and causes the formation of pedicles.

Our observation in many of these cases convinces us that this description of the formation of "inflammatory" polyps is adequate. Grossly (figs. 1 and 2) these resemble closely the adenomatous polyps of the second group, but the dark discoloration so frequently seen in the serrated portion is absent. Microscopically the polyps are in all stages of chronic inflammation, and in the serrated portion are numerous nests or groups of glands seemingly caught in a dense inflammatory meshwork. The difference between these and the ordinary adenomatous polyps is the great increase in inflammatory and fibrous tissue in the polyps, yet multiple carcinomas have been

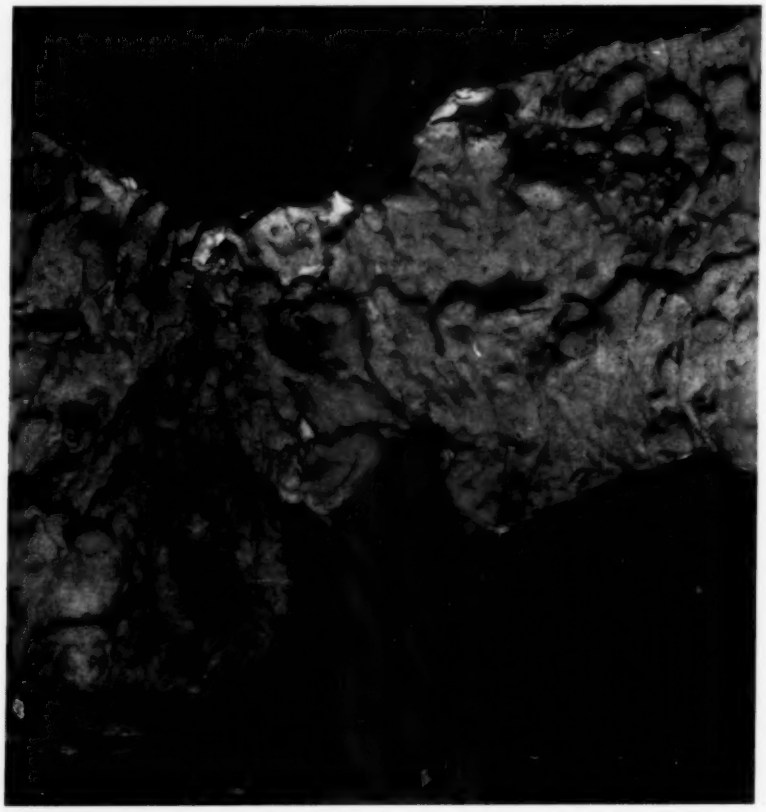

FIG. 1. Portion of a colon removed at necropsy. The polypoid tags of mucosa are shown.

FIG. 2. Portion of a colon in which the chronic ulcerative colitis has healed, leaving disseminated polyps.

demonstrated in these hyperplastic areas of hypertrophy. This raises the question of whether or not these polyps are prone to malignant change.

The ratio of these to the other polyps of multiple polyposis is as 4:1, that is, of every five cases of multiple polyps observed at The Mayo Clinic, four will be on a basis of chronic ulcerative colitis. With this large disproportion of the two types, it seems important to determine whether the treatment for both should be the same. The answer to this will depend on many factors, among them, the general condition of the patient, the extent of disease, the amount of destruction of the large intestine by colitis, the activity of the colitis, and the length of time the patient has had the trouble.

The symptoms of polyposis, the result of chronic ulcerative colitis, vary little from those of actual colitis. A remission of the disease may have started and the patient gradually notices a return of bleeding, more tenesmus, frequency, and sometimes blood out of proportion to the seeming severity of other symptoms. Further proctoscopic examination reveals numerous polyps in the rectum and sigmoid of all sizes and shapes. Almost invariably they occur as healing or improvement takes place. In several instances cases of early fulminating chronic ulcerative colitis have been ob-

served from the onset, through a remission, and to the stage of polypoid change. The following case histories are illustrative:

Case 1. A man, aged twenty years, was admitted to The Mayo Clinic September 26, 1925. He had had attacks of bloody dysentery, with a maximum of thirty to forty stools in twenty-four hours, for nineteen months; sometimes the attacks lasted for a hundred days in succession. A diagnosis of amebic dysentery had been made although amoebae had not been found. He was acutely ill, with a maximal temperature of 102°F. The stools were mixed with pus and blood. Incontinence was troublesome. He had lost 24 pounds.

The pulse was 90; the blood pressure was 120 systolic and 90 diastolic, measured in millimeters of mercury. The abdomen was moderately tender. Proctoscopic examination disclosed diffuse, granular involvement of the mucosa of the rectum and sigmoid, edema, a tendency to hemorrhage, and scattered punched-out ulcers. A diagnosis of chronic ulcerative colitis was made. During the next five months the patient's condition fluctuated. Improvement was slow but by October 24, 1925, he was well enough to be dismissed from observation.

The patient returned in February, 1926, more seriously ill than before. He had failed steadily during the month. Proctoscopic examination showed large, sloughing, ragged, undermining ulcers of the rectum with bridging of the mucosa between large ulcers. Ileostomy was suggested as an emergency, but because of the patient's condition it was not done. Treatment then consisted of tincture of iodine by mouth, large doses of kaolin alternating with bismuth, opium and paregoric and small doses of vaccine prepared from the diplostreptococcus which had been isolated in practically pure culture from the ulcers in the rectum on both visits to the clinic. The patient began to improve slowly and after several months was able to go home.

The patient returned again in September. He had gained 37 pounds in the preceding four months. He was having six to seven bowel movements in twenty-four hours; the stools occasionally contained a little blood and mucus. Proctoscopic examination showed the mucosa to be practically normal; a few pitted scars were scattered over a slightly pale mucosa. There were multiple polyps from 0.3 to 0.7 mm. wide and from 0.3 to 1.5 cm. long; some of these bled easily. Polyposis had followed the healing of advanced chronic ulcerative colitis. Clinically the patient was in excellent condition. He was dismissed with instructions to take vaccine subcutaneously. In May, 1927, he reported that he had had the best winter since the beginning of his illness. He had gained 50 pounds and looked the picture of health. He had averaged three bowel movements daily for months and had not seen blood in his stools for at least a month. Proctoscopic examination now showed that the chronic ulceration of the colon was healed, leaving polypoid areas and polyps. Certain small polyps that were seen in January had disappeared. The mucosa between the polyps was normal except for the scars of the infection. A series of treatments by fulguration of the rectal polyps was carried out without incident. The patient was free from symptoms of bowel trouble but because all of the polyps could not be fulgurated at the first visit, he returned in December, 1927, at which time the proctoscopic examination disclosed several polyps still in the rectum but the mucosa was normal. The polyps were again fulgurated. In August, 1928, there were some scars in the rectum but no ulceration; the lumen was practically normal in diameter, and the areas fulgurated were free from polyps.

Case 2. A man, aged twenty-nine years, came to the clinic February 22, 1927, with a history of bowel trouble dating back to his service in the United States Army in 1918. The trouble started as an acute fulminating type of dysentery which slowly improved after he was dismissed from the service but he had never been entirely relieved of his trouble. There had always been blood and mucus in the stools. Amoebae or other parasites had not been found.

On the patient's admission he was having between five and eight rectal discharges in twenty-four hours, and otherwise he felt

well; his weight was about normal. Gastric acids were 60 and 40, total and free, respectively, with 130 c.c. of gastric contents after a test meal. The Wassermann reaction of the blood was negative.. Urinalysis was essentially negative on two occasions. The hemoglobin was 80 per cent, erythrocytes numbered 4,640,000, and leucocytes, 7,000. Three examinations of the stool on three successive days failed to show parasites or ova. There was a very small tonsillar tag which did not appear to be significant. A roentgenogram of the stomach was negative; one of the colon showed the deformity of chronic ulcerative colitis of the descending colon and sigmoid. The proctoscopic examination gave the typical picture of chronic ulcerative colitis, graded 2, with contraction to about a third the usual dimension of the rectum. Treatment consisted of the subcutaneous injection of a filtrate of the diplostreptococcus of chronic ulcerative colitis, and some kaolin and bismuth by mouth from time to time. Local rectal instillations of witch-hazel were also given. The patient was dismissed, April 29, improved. He returned, August 22, with the ulceration definitely improved, but definite evidence of disease and numerous small polyps in the rectum and sigmoid were present. Injections of the vaccine and filtrate were more or less continuous thereafter, with only the usual remissions which are allowed between. April 1, 1929, the urinalysis and blood count were within normal limits. A roentgenogram of the teeth was negative. A roentgenogram of the colon showed the deformity of chronic ulcerative colitis distal to the splenic flexure, with defects resembling polyps in the sigmoidal portion of the colon (figs. 3 and 4). On proctoscopic examination the activity of the disease was graded 1. Numerous rectal and sigmoidal polyps, which were slightly larger than at the former examination, were present. April 5, the polyps in the rectum and sigmoid within reach of the proctoscope were fulgurated. Further local treatment was instituted, including the instillation into the rectum of witch-hazel, alternating with mercurochrome. The patient was dismissed May 8, improved.

Late in June, a roentgenogram taken elsewhere was referred to the clinic. The plates showed defects similar to those in the roentgenogram made in April, which suggested that there were polyps in the sigmoid beyond the reach of the proctoscope.

Case 3. A man, aged forty-four years, came to the clinic June 27, 1925, with a three-months' history of diarrhea. The trouble started with a slight increase in bowel movements, followed by marked constipation with severe abdominal cramps for a few days. After a laxative had been given, rectal discharges containing blood and pus were frequent; they were accompanied by severe abdominal pain. Three weeks before admission there had been an average of between ten and fifteen bloody, purulent rectal discharges in twenty-four hours. He had lost 30 pounds during the month before admission.

When the patient was admitted he was having an average of fifteen to twenty rectal emissions with much cramping and tenesmus. His facial expression was anxious. Proctoscopic examination revealed chronic ulcertative colitis, graded 3. The disease appeared to be in the acute stage; the rectum had a meaty appearance, and the bowel was contracted to about half the usual dimension. A roentgenogram of the thorax was negative. The hemoglobin was 50 per cent; erythrocytes numbered 3,080,000 and leucocytes 8,200. The Wassermann reaction of the blood was negative. Repeated examinations of the stool failed to reveal parasites or ova. The patient was too sick for examination by barium enema and this was not undertaken until July 28; it resulted in a diagnosis of chronic ulcerative colitis of the entire colon. A culture of the ulcers in the rectum yielded the diplostreptococcus which usually is found in these cases. Treatment was begun with vaccine filtrate. Improvement was slow but progressive. August 25, the proctoscopic examination disclosed 90 per cent improvement of the rectum. The patient was dismissed and advised to continue with vaccine treatment at home. He was examined May 11, 1926. He had improved steadily during the year and the proctoscopic examination at this time showed the activity of the disease to be of grade

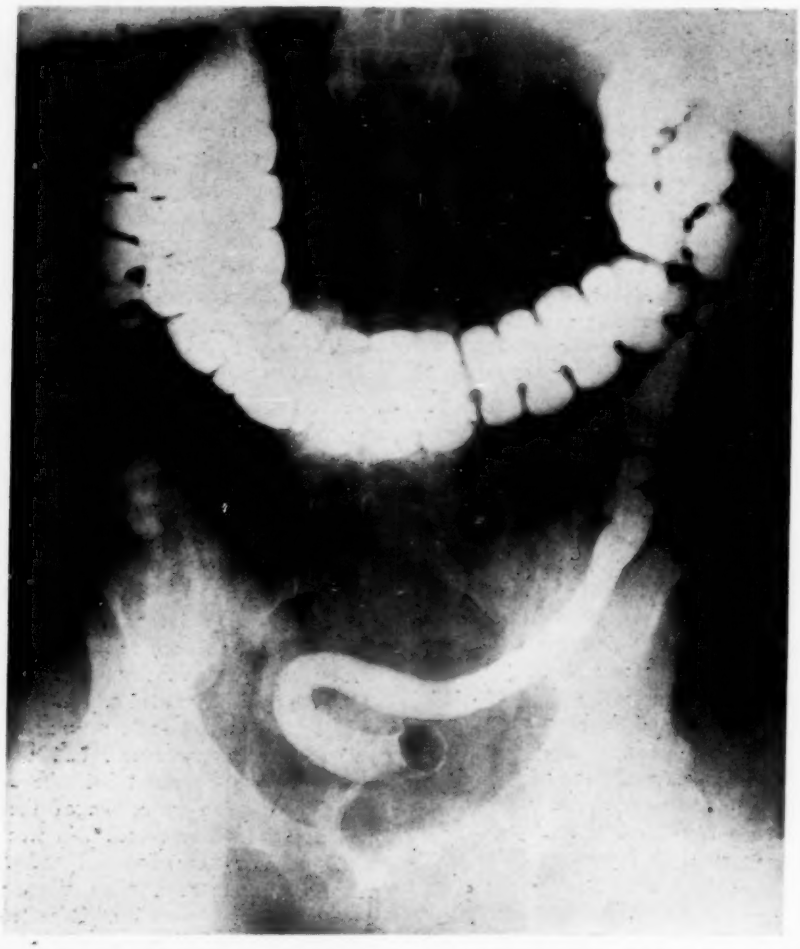

FIG. 3 (case 2). Roentgenogram of colon by barium enema. Deformity of chronic ulcerative colitis, in February, 1927.

1 or less, and it was noted that most of the rectum was covered by normal-appearing mucosa. There was one small polyp, about 8 mm. high, 4 cm. above the anus. He was free from symptoms. Early in January, 1927, there was a slight recurrence of the trouble, and examination, January 26, showed increased activity of the disease, graded 2. The polyp had increased to twice the size at the first examination. Improvement again followed on similar treatment and the next visit, in October, 1928, showed only a residue of the disease. Roentgenologic examination of the colon by barium enema on the various admissions was as follows: August, 1925, extensive chronic ulcerative colitis with much destruction of mucosa; May, 1926, residual ulcerative colitis; April, 1927, evidence of polyposis in the transverse and descending colon, and the return of many haustra, and October, 1928, old chronic ulcerative colitis, and polyposis of the descending colon (figs. 5, 6 and 7). Because of the severity of the disease and the nature of the polyps, operation was not undertaken. January 1, 1930, the patient was in good

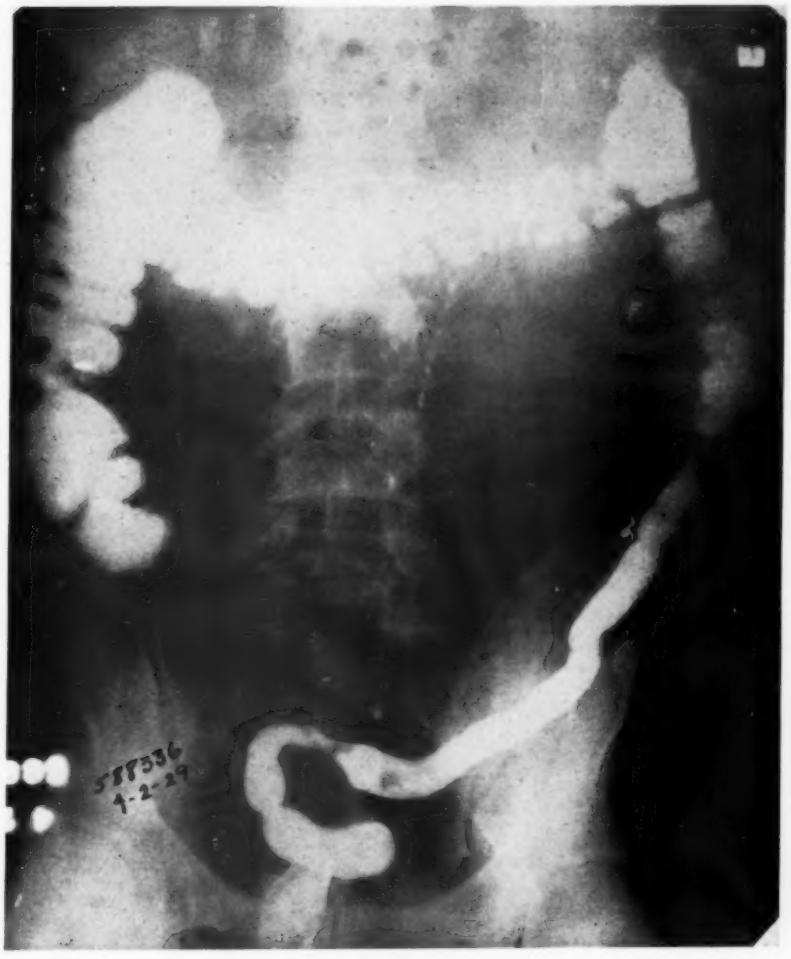

FIG. 4 (case 2). Roentgenogram of colon by barium enema. Defects in the sigmoid corresponding to polypoid lesions in the presence of the deformity of chronic ulcerative colitis.

condition. He had had the specific vaccine at intervals during this time.

As in other large intestinal lesions, the roentgenogram and the proctoscope are the most valuable means of studying these polyps in the living subject. Biopsy often lends valuable aid in diagnosis. The proctoscope gives little evidence of the extent of the polyposis. Here the roentgen ray offers the best hope of accurate information. Although proctoscopic examination has greatly advanced knowledge of benign lesions of the colon, repeated instances may be noted in which, in spite of most careful efforts and by the use of all known methods, it has not been possible to obtain information of much value in some cases of polyposis of the colon. In view of this, Fischer in-

jected a thin medium by rectum and then filled the colon with air. He stated that one may thus see the polyps as a spotted condition on a diffuse base. Eickenbusch was able to confirm this observation. Such slight filling with the ordinary contrast medium, he stated, gave a good picture.

Our experience, however, suggests that the best method at present is the use of the barium enema. Occasionally in the cases of severe ulcerative colitis with the deep ulcers, numerous small filling defects may be seen along the margin of the intestine, not unlike the defects of polyposis, but careful observation will show distinguishing features. The edge of the colon in the case of polyposis is usually smooth, and the rounded defects are disseminated throughout the extent of the colon (case 3, figs. 5, 6 and 7).

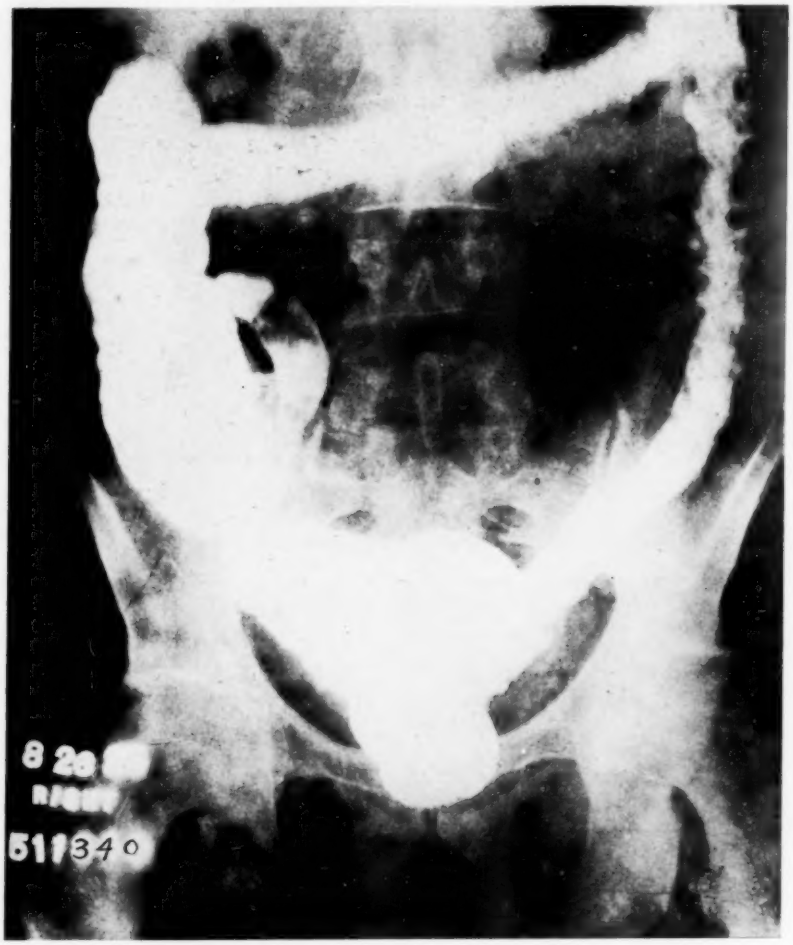

FIG. 5 (case 3). Roentgenogram of colon by barium enema. Severe extensive disease with much destruction of mucosa, in August, 1925.

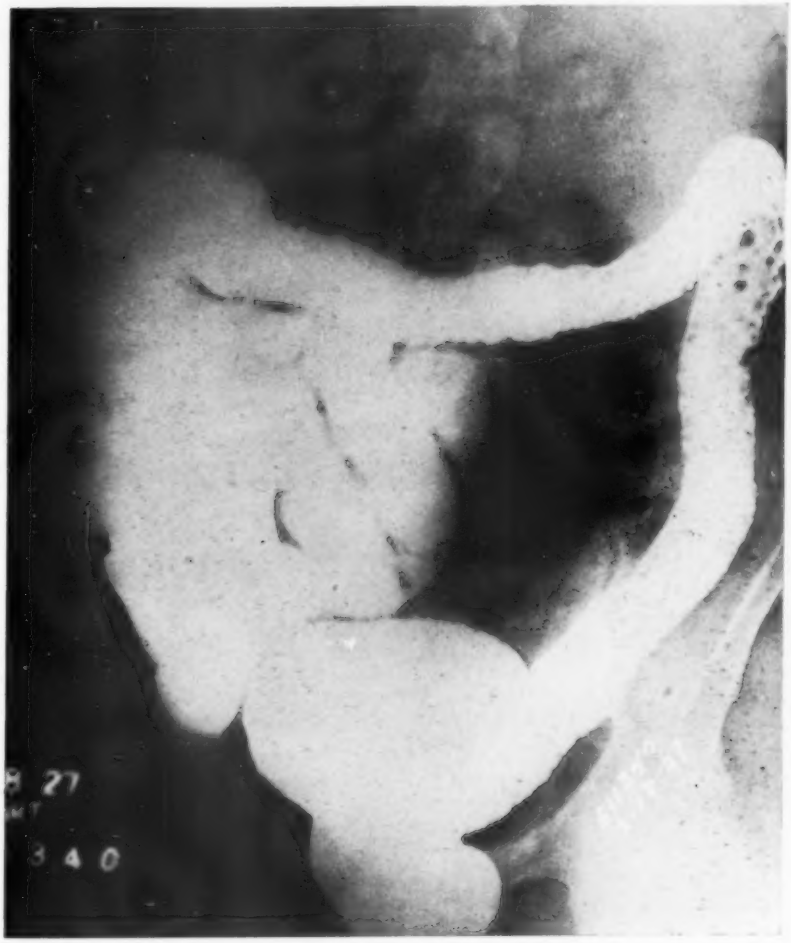

FIG. 6 (case 3). Roentgenogram of colon by barium enema. Beginning smoothing out of edge of colon with numerous rounded defects, in February, 1927.

Polyposis in the presence of chronic ulcerative colitis may offer a grave prognosis. We are engaged at present in a microscopic study, tracing the sequence of events through the ulcerative phase to the formation of polyps, and attempting to determine the nature of these polyps. A few of the patients, like those in the cases reported, are pursuing their usual activities, and are feeling well, and yet we know that the lining of their intestines contains polyps. We have fulgurated, as a routine, all the rectal polyps in these cases. The risk of surgical manipulation of the wall of the colon once impregnated with the residue of chronic ulcerative colitis is high. It is hoped that future work will make possible a definite prognosis in this type of polyposis.

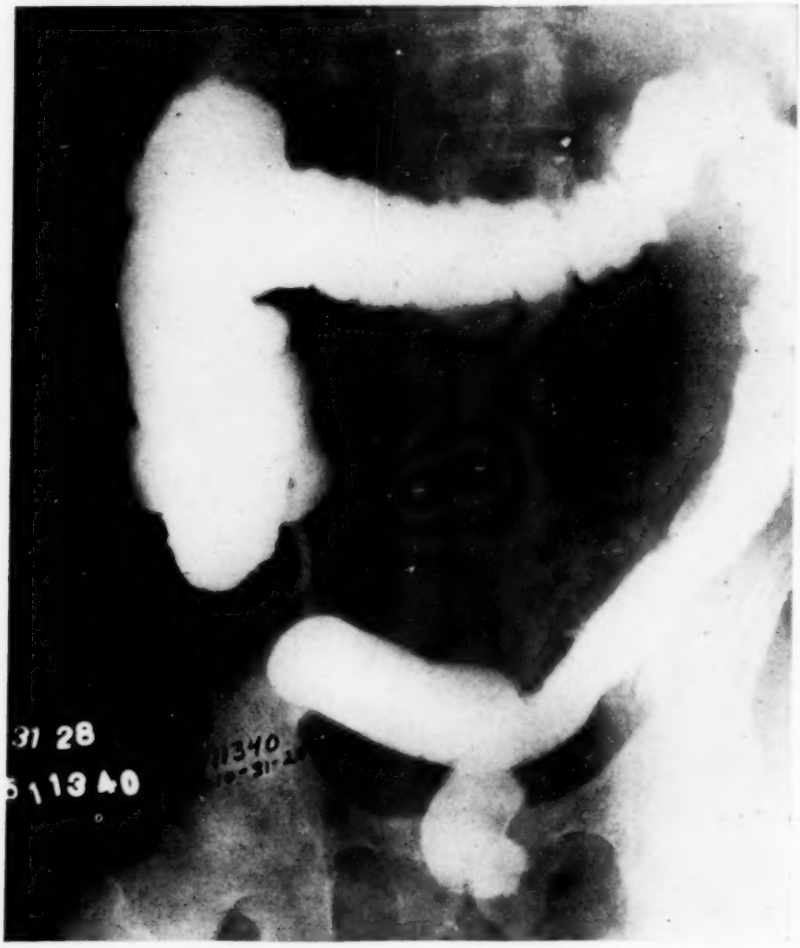

Fig. 7 (case 3). Roentgenogram of colon by barium enema. Return of haustra but presence of numerous rounded defects, in October, 1928.

Comment

Polyposis of the large intestine is present in 10 per cent of cases of chronic ulcerative colitis. It occurs after severe colitis or in long-standing, slowly progressing colitis in which there is a tendency to recurrent severe exacerbations of the disease. The polyps contain large amounts of inflammatory tissue. Proctoscopic examination is the best means of diagnosis in most cases. Roentgenologic examination gives valuable information in many cases. We have had an opportunity to see patients in severe exacerbations of bacterial chronic ulcerative colitis, with formation of polyps later, and finally with malignant disease. Studies are under way to trace the formation of the polyps and their subsequent course in a large series of specimens obtained at operation and at necropsy.

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Observations on the Etiology of Gall Stones*

By E. L. Walsh, M.S., and A. C. Ivy, Ph.D., M.D.,
(Department of Physiology and Pharmacology, Northwestern University Medical School), Chicago, Illinois

THE most accepted general cause of the origin of gall stones is bile stasis. Beyond this, views vary widely. Naunyn[1] believes that a second cause is an ascending infection which causes "the stone-forming catarrh of the vesical mucosa," the cholesterin and calcium of the stone coming from the inflamed mucosa. Although he admits that some cholesterin may come from the bile, he attaches no great significance to diet nor to changes in cholesterin metabolism. Aschoff and Bacmeister[2] classify gall stones etiologically into two general divisions, (a) the non-inflammatory or metabolic, and (b) the inflammatory, believing that the origin of the cholesterin stone is due first to excessive cholesterin in the bile, plus concentration and stasis of bile in the gall-bladder. In contradistinction to these views it has been suggested recently by Halpert[3] on the basis of his opinion that the bile which enters the gall-bladder never leaves it, that gall stones are due to the failure of the inflamed gall-bladder to reabsorb cholesterin.

The gall stone problem attracts the physiologist, first, because if stasis is a factor, the physiologist wants to know the cause of the stasis; and second, if cholesterin metabolism is at fault, he wants to know how this affects the bile and its relation to the other biliary constituents. Our immediate interest in the problem was attracted by two reports (4, 5, 6,) in the literature, one being that human gall stones of the mixed cholesterin variety when placed in the gall-bladder of the dog disappear in several months, the second being that cholesterin stones have not been produced experimentally in the dog.

We first desired to convince ourselves that human gall stones would dissolve when placed in the dog's gall-bladder, and then to ascertain if we could influence by diet and other procedures the rate of solution, and then to study why the gall stones are dissolved, hoping that our results might throw some light on the etiology of gall stones in man and be of value in the prophylactic treatment of this disease.

Human gall stones are dissolved when placed in the gall-bladder of the dog: Human gall stones of the mixed cholesterin-calcium-pigment type were obtained through the courtesy of various Chicago surgeons, washed with sterile salt solution, weighed, and

*Presented before the American College of Physicians, February 10, 1930, Minneapolis, Minn.

placed in the gall-bladder of dogs on a diet of yellow corn-meal, bread and bone soup. The rate of disappearance was followed by use of the method of gall-bladder visualization. When the stone became quite small the stone was removed and weighed.

It was found that the stone began to lose weight within three days and in the course of from 65 to 156 days would lose from twenty to ninety-six per cent of its weight. (Table 1). If, however, a definite chronic fibrous cholecystitis followed the introduction of

TABLE 1. SHOWING SOLUTION OF HUMAN GALL STONES IN GALL-BLADDER OF THE DOG.
No Solution in Peritoneal Cavity. Stock Diet.
Ten stones in gall-bladder 65 days; 4 longer than 65 days.
The capital letter indicates the human source of the stone.

Stone Number	Days in Gall-bladder	Original Weight	Final Weight	Loss Weight	Percent Loss	Remarks
	139	2.014	0	2.014	100.0	
	156	1.000	0.074	0.926	92.6	Followed by X-ray
	113	0.896	0.111	0.785	87.5	Followed by X-ray
3 P 10	144	1.395	0.058	1.337	96.0	Followed by X-ray
3 J	65	1.124	0.141	0.983	87.5	
2 J	65	1.551	0.883	0.668	44.5	
7 K	65	1.535	0.272	1.263	82.2	
1 Y	114	0.762	0.117	0.645	84.6	
2 Y	65	1.353	0.429	0.924	61.0	
3 Y	65	2.348	2.276	0.072	3.0	Chronic*Cholecystitis
1 B	65	3.148	2.366	0.782	24.8	
	65	1.710	1.051	0.659	38.5	
1 G	65	1.286	0.359	0.925	72.0	
12 G	65	1.118	0.586	0.532	43.7	
1 S	65	3.346	3.193	0.153	4.4	Chronic*Cholecystitis
In Peritoneal Cavity						
12 G	65	1.320	1.313	loss 0.007	—0.5	
5 X	65	1.642	1.656	gain 0.014	—0.8	

*Bile thin, low sp. gr. Note that stone did not dissolve much.

The average amount dissolved in the eight stones in normal gall-bladder for 65 days is 56.7 per cent, the maximum being 87.0 per cent, the minimum 24.8 per cent.

Stock diet consists of yellow corn meal, bread, and bone soup.

the stone, the stone lost only a small percentage (3-5%) of its weight, in which condition light yellow bile of low sp. gr. was found in the gall-bladder. This observation confirmed the findings of Harley and Barratt.[4] When gall stones were placed in the peritoneal cavity a material change in weight did not occur.

For purposes of study it was necessary to fix a uniform period of time to leave the stone in the gall-bladder. This was fixed for a 65 day period. The next factor that had to be considered was that different stones might dissolve at a different rate. On putting stones from the same patient into the gall-bladder of different dogs, or even on putting the same stone in the gall-bladder of different dogs, it was found that the rate of solution varied definitely in the different dogs, which rendered it valueless to study or control this factor.

In eight dogs on the stock diet, the average amount of the stone dissolved in 65 days was 56 per cent, the maximum being 87 per cent and the minimum 24 per cent.

Effect of olive oil added to stock diet: In a series of dogs 60 cc. of olive oil was given the dogs, two hours before the usual stock-diet meal, to ascertain if the olive oil might have an effect on the rate of solution. The average amount dissolved in 10 dogs of this regime was 53 per cent; the maximum being 86 per cent and the minimum 21 per cent. The figures do not show that the olive oil had any influence. (Table 2).

Effect of cocoanut oil added to stock diet: Because it is stated that the people in Siam and Java only rarely have gall stones and their chief source of fat is cocoanut oil, it was decided to use this fat. Eighty cc. of cocoanut oil was given daily two hours before the stock-diet meal. The average amount of stone dissolved in 10 dogs in this series was 60 per cent, the maximum being 100 per cent and the minimum 35 per cent. Although this is 4 per cent more than the control group, we believe that it is not significant. (Table 3).

Does the gall-bladder of the dog secrete something which dissolves the stone? This obviously is not an easy question to answer. In order to obtain some evidence on this question, we tied the cystic duct of five dogs, emptied the bile from the gall-bladder and introduced the stone. From 65 to 70 days later the stone was removed. Two of the five had gained from 4 to 5 per cent in weight, one was unchanged, and two lost 5 per cent. All of these gall-bladders showed pathological changes. Because of the nature of these results the question remains unanswered. Further, experiments of a direct nature are under way. However, data given later in this paper indirectly indicates that the gall-bladder does not secrete a solvent, and that the solution of the stone is caused by the bile.

Effect of ligation of cystic duct on the dog's gall-bladder: Because of the pathological changes that resulted following ligation of the cystic duct with a stone in the gall-bladder, we decided to ascertain the effect of ligation of the duct alone. The cystic duct was tied in four dogs and the gall-bladder ex-

TABLE 2. SHOWING SOLUTION OF HUMAN STONES IN THE GALL-BLADDER OF THE DOG. OLIVE OIL IN DIET

60 cc. of olive oil per day 2 hours before meal by stomach tube.

Stone Number	Days in Gall-bladder	Original Weight	Final Weight	Loss Weight	Percent Loss	Remarks
10	65	1.292	0.178	1.114	86.8	
3	65	2.569	0.911	1.658	64.5	
4	65	3.085	1.865	1.220	39.6	
2	67	2.943	2.319	0.624	21.1	Adhesions-Moderate Cholecystitis
4 X	65	2.039	0.365	1.674	82.3	Pregnant
10 G	65	1.119	0.727	0.392	35.0	Slight Cholecystitis
7	65	1.806	1.187	0.619	34.2	Slight Cholecystitis
2 CC	66	1.176	0.557	0.619	52.5	
13	65	2.853	1.463	1.390	48.7	Slight Cholecystitis
1	65	3.643	3.319	0.324	8.8	Marked Cholecystitis-Adhesions
1 Y	65	2.130	0.605	1.525	71.6	

The average in 10 dogs, omitting Stone 1 because of the marked cholecystitis, is 53.6 per cent, maximum 86,8, minimum 21.1.

Compare with Table 1, noting that cholecystitis is apparently more frequent in this series.

amined two months later. In one the gall-bladder had been replaced by fibrous tissue. In the others the gall-bladder wall was thickened and the mucosa abnormal. The gall-bladder in each case contained a colorless or light brown viscid secretion and small flecks or concretions of pigment and carbonates, the largest concretion weighing 0.184 g.

Effect of stricture of common bile duct on rate of solution of human gall stone: A stricture of the common bile duct was produced in four dogs by inserting a glass cannula having an inside bore of 1 mm. into the common duct. This produced a stasis of bile which was demonstrated by the fact that the bile ducts above the point were dilated and from 50 to 100 mgm. of "flaky" sediment of the pigment and carbonate variety was found in the gall-bladder when the dogs were autopsied from 61 to 70 days later. The average loss of weight of the stones was 44 per cent which is less than that of the control dogs without stricture on the same stock diet. This is not as marked an effect as we had anticipated. Our explanation will be offered later. Further experiments on this

TABLE 3. SHOWING SOLUTION OF HUMAN STONES IN THE GALL-BLADDER OF THE DOG. COCOANUT OIL IN DIET.
80 cc. of cocoanut oil per day 2 hours before meal by stomach tube.

Stone Number	Days in Gall-bladder	Original Weight	Final Weight	Loss Weight	Percent Loss	Remarks
3 Y†	65	1.072	0.245	0.727	67.6	Followed by X-ray
100*	65	2.488	1.604	0.884	35.4	Marked Cholecystitis
7	65	1.167	0.211	0.976	82.3	
OSM	65	4.709	3.948	0.761	16.3	Moderate Cholecystitis Stone adherent to GB in several small areas
12	66	2.827	1.077	1.750	62.0	
1 GO	67	1.543	0.945	0.598	38.4	Moderate Cholecystitis
3 X	65	2.019	0.887	1.132	56.0	
4 CC	68	1.147	0.596	0.551	49.1	
10 CC	65	1.194	None	1.194	100	Lymphoid-Hyperplasia marked
1 BG	67	1.457	0.825	0.632	43.3	
13 CC	50	0.982	0.319	0.663	71.5	

†This dog is the same as 1 B in Table 1. Note: Dissolved 67 per cent the first time and 24 the second time.

*This dog is the same as 1 G in Table 1. Note: Dissolved 72 per cent the first time, 35 per cent the second time with cholecystitis.

The average for this group, omitting dog OSM and dog 13 CC, nine dogs, is 59.3 per cent, maximum 100 per cent, minimum 35. Including dog 13 CC, average is 60.5 per cent.

question are in progress in which the procedure is modified. (Table 4.)

Effect of stricture of common bile duct on gall-bladder of the dog: A stricture of the common bile duct was produced in seven dogs by the method used above. (Table 5.) Two of the animals died in 49 and 62 days respectively, one of empyema of the gall-bladder and multiple liver abcesses and the other of bile peritonitis due to a small opening (0.5 mm. diameter) in the common duct. The others were killed from 62 to 80 days after the operation. The ducts of all the dogs were dilated and sediment in amounts varying from 50 to 100 mgm. was found in the gall-bladder as well as in the dilated common duct above the cannual. A hyperplasia of the lymphoid tissue and of the mucosa of the gall-bladder was found in three of the five dogs. In the other two the mucosa was normal. These results along with those just mentioned above show that stasis produced by this method may cause changes in the histology of the gall-bladder mucosa and

TABLE 4. EFFECT OF STRICTURE OF COMMON BILE DUCT ON SOLUTION OF HUMAN STONE IN DOG'S GALL-BLADDER
Stock Diet

Dog Number	Stone Number	Days in Gall-bladder	Original Weight	Final Weight	Loss Weight	Percent Loss	Remarks
143	5 CC	69	1.178	0.813	0.365	44.9	Pericholecystitis
167	A P	64	0.830	0.224	0.606	74.2	Gall-bladder normal
173*	P R	61	2.019	1.486	0.533	26.4	Bile very thick
140	1 M	70	1.147	1.000	0.497	33.1	Gall-bladder normal Lymphoid Hyperplasia

*Round worm in gall-bladder 2 inches long. In all these dogs there was 50 - 100 mgm. of flaky sediment of the nature of calcium-pigment and X-CO_3, which is abnormal for the dog's normal gall-bladder. Average 44%.

TABLE 5. EFFECT OF STRICTURE OF COMMON BILE DUCT ON GALL-BLADDER OF THE DOG.

Dog Number	Tome of Observation Days	Findings
118	62	Bile peritonitis. Slight cholecystitis. Dilation of ducts. Sediment in gall-bladder.
188	80	Killed. Gall-bladder normal. Ducts dilated. Sediment in gall bladder.
134	64	Killed. Gall-bladder thin wall. Normal mucosa. Bile very thick with mucin. Ducts dilated. Sediment in GB. Suspended in bile.
144	67	Killed. Lymphoid hyperplasia. Sediment in gall-bladder. Ducts dilated.
145	68	Killed. Lymphoid hyperplasia. Bile very thick and flaky with sediment. Ducts dilated.
163	49	Multiple liver abscesses. Empyema of the gall-bladder.
165	67	Killed. Hyperplasia of mucosa. Ducts dilated. Sediment in gall-bladder.

Sediment was found in every gall-bladder either in the mucous along the mucosa or suspended in the bile. The sediment varied from 50 to 100 mgm. in amount and was of the calcium-pigment and X-CO_3 variety.

lymphoid tissue and in the character of the bile.

Effect of chronic reverse duodenal peristalsis on gall-bladder: The above method of producing stasis was anatomical and rarely occurs in life. Since duodenal motility is concerned in controlling the flow of bile from the biliary passages, it was thought that by permanently altering duodenal motility we might cause a biliary stasis. In order to permanently alter duodenal motility a two inch loop of duodenum was excised and turned end-for-end and sutured, or a reversed duodenal loop was made. In the duodenum then there existed a continuous reverse peristalsis leading to duodenal stasis and abnormal motility. In the course of eight months this leads to a marked hyperplasia of the gall-bladder mucosa and lymphoid tissue, (Dr. L. A. Crandall and E. L. Walsh) and on autopsy the bile is found to be thick and contains sediment of the pigment and carbonate type.

In this type of experiment, in which the disturbance is more of the nature of perverted physiology, one obtains the same results as in the mechanical or anatomical stricture, and reverse peristalsis in the duodenum is known to occur in nausea, vomiting and in the morning sickness of pregnancy.[7 8]

Experimental chemical acute cholecystitis and rate of solution of gall stones: Acute chemical cholecystitis was produced in three dogs each with alcohol and Dakin's solution. The results showed that the cholecystitis had to be diffuse and considerable connective tissue proliferation present before the rate of solution of the stone was affected. Three dogs, in which there was marked hyperplasia of the mucosa, hyperplasia of the lymphoid tissue and slight fibrosis, dissolved from 59 to 100 per cent of the stone in from 55 to 70 days.

It was evident from these experiments and the observations recorded above that if the gall-bladder could concentrate bile the stone would be dissolved rapidly, but if it could not concentrate the bile very little of the stone would be dissolved.

In Vitro Experiments

The observations above indicate that there is something in dog's gall-bladder or concentrated bile which is responsible for dissolving the stone.

It has been found that ox bile, [10] dog's bile,[10] soap solution,[9] have a solvent action on human gall stones. It is claimed that sodium taurocholate and glycocholate have a slight solvent action and that sodium desoxycholate has 3 or 4 times the action of the other bile salts.[11]

Tre results of our in vitro experiments are shown in Table 6.

It is to be noted, (1) that we failed to find the two chief bile salts (Merck) present in bile to possess a solvent action; (2) that human bile caused no solution; (3) that dog's gall-bladder bile caused solution and (4) that when diluted dog's gall-bladder bile or the dog's hepatic bile was used no solution of the stone occurred. This confirms our in vivo observation that the concentration of the bile by the dog's gall-bladder is essential for solution of the human gall stone. It also shows that the lower concentration of cholesterin in dog's bile than in human bile

TABLE 6. EFFECT OF VARIOUS SOLUTIONS ON GALL STONES WHEN SHAKEN IN VITRO FOR 15 DAYS AT 37°C.

Solution Used—Solution Changed Daily Solution Agitated	No. of Expts.	Av. % Loss	Av. % Gain	Max. % Loss	Max. % Gain	Min. % Loss	Min. % Gain
Distilled water	2		0.3		0.7	0.1	
0.9% Sodium chloride in water	4	0.1		0.2			0.0
1% Sodium glycocholate in water	4		0.1		0.4		0.0
1% Sodium taurocholate in water	4		0.2	0.3	0.6		
Human fistula bile	4	0.1		0.7	0.2		
Human gall-bladder bile	8		0.1	0.3	0.7		
Dog's fistula bile	8	1.6		4.3		0.4	
Dog's gall-bladder bile	12	28.8		82.0		11.8	
Dog's gall-bladder bile diluted 10 times with water	5		0.5	0.5	2.6		
1% Butyric acid in pH 6 buffer solution	4		11.1		29.8		0.5
1% Butyric acd in pH 8 buffer solution	4		8.3		14.0		0.5
1% Oleic acid in pH 6 buffer solution	18	13.6		41.1		5.8	
1% Oleic acid in pH 8 buffer solution	18	25.1		72.5		4.2	
1% Myristic acid in pH 8 buffer solution	11	27.4		41.7		12.1	
1% Lauric acid in pH 8 buffer solution	13	74.8		100.0		50.6	
Solution Changed Every 3 Days							
1% Myristic acid in pH 8 buffer solution	5	10.9		14.1		5.6	
1% Lauric acid in pH 8 buffer solution	7	29.5		39.8		20.0	
Solution Changed Every 3 Days—No agitation							
1% Lauric acid in pH 8 buffer solution	2	7.6		8.5		6.7	

is not the factor, because if it were a factor, then the stone should dissolve faster in the more dilute hepatic bile of the dog than in the more concentrated gall-bladder bile.

The various fatty acids shown in Table 6 were examined for their solvent action because we thought that the fatty acids and soap fraction of the bile which is higher in the dog than in man, might be the important factor. To our surprise the stones gained weight in the butyric acid solution, but, however, lost weight in oleic acid, the chief fatty acid in olive oil, and in myristic and lauric acid, the chief fatty acids in cocoanut oil. The solvent action of lauric acid is to be especially noted. It is also interesting that the pH of the fatty acid solution is also

important in that there is more of the soap of the fatty acid present in the pH 8 buffer solution than in the pH 6 buffer solution.

Table 7 shows that stones from different patients differ in the rate at which they lose weight, but the difference is not as marked as we had anticipated.

Chemical Analysis of Bile

Since soap from the above results has such a solvent action on human gall stones of the cholesterin-pigment-calcium variety, studies on the relative amounts of cholesterin on the one hand and the saponifiable substances on the other in human and dog's bile was undertaken.

The data obtained are shown in Table 8. In the dog the ratio of saponifiable substances to cholesterin is approximtely 20 to 1, whereas in man it is only approximately 2 to 1.

Our results indicate that the soap-cholesterin ratio in bile is very important and that an optimum concentration of soap is necessary for the solution of the human gall stone. Soap is the best solvent we have yet used which is confirmatory of the observation of Brockbank.

We have attempted to determine the soap content of human and dog's bile, but have been unable to obtain a method that would yield accurate results. The older values of soap in the bile, we believe, are unreliable.

Conclusions and Summary

1. Human gall stones of the mixed cholesterin variety are dissolved when placed in the gall-bladder of the dog, confirming Harley and Barratt,[4] Labes[5] and Harrison and Barber.[6]

Table 7. Showing that the Rate of Solution of Stone Depends to Some Extent on the Stone

Averages are taken from daily weighing of stone until completely dissolved. The latter indicates that the stones were from the same patient. In vitro experiments with lauric acid in pH 8.0 buffered solution, solution changed daily.

Stone Number	Original Weight	Days for Complete Solution	Average Loss per day in mgm.
1 L	0.830	34	24
2 L	0.962	35	27
1 M	1.182	38	31
2 M	1.232	40	30
1 P	1.153	27	43
2 P	1.512	39	39
1 Q	0.924	38	24
2 Q	1.085	35	31*

*Some fragmentation occurred which could not be checked by weight and accounts for the discrepancy.

TABLE 8. RESULTS OF CHEMICAL ANALYSIS OF HUMAN AND DOG GALL-BLADDER AND FISTULA BILE.
Saponifiable and Non-saponifiable Fractions.
Saponifiable fraction weight checked by titration.

GALL-BLADDER BILE

Case Number	Human Post-mortem Bile		Dog	
	Non-saponifiable Cholesterin	Saponifiable Fat, fatty acid, Soap, lecithin	Non-saponifiable Cholesterin	Saponifiable Fat, fatty acid, Soap, lecithin
1	0.358	2.066	0.188 0.140	4.016 3.544
2	0.972 1.296	1.104 1.440	0.106 0.108	4.428 4.488
3		1.136 1.148	0.086	4.220 4.294
4	0.602 0.578	1.670 1.692	0.136	3.740 3.754
5	0.162 0.134	0.684 0.619	0.354 0.356	3.926 3.858
6			0.108 0.102	3.920 3.938
7			0.132 0.130	4.268 4.172
Average	0.586	1.284	0.162	4.040

FISTULA BILE

Case	Human	Cholecystostomy	Dog	Common Duct Gall-bladder removed
1*	0.162 0.179	0.514 0.526	0.019 0.015	0.485 0.452
			0.020 0.032	0.495
			0.017 0.017	0.259 0.257
			0.032 0.030	0.094 0.160
Average	0.171	0.520	0.020	0.314

*Probably concentrated 2 to 4 times by passage through gall-bladder.

2. A diffuse fibrous cholecystitis prevents the solution of the gall stone, which is due to the failure of such a gall-bladder to concentrate the bile. Concentration of the dog's bile is necessary for solution of the gall stone.

3. We were unable to show that the addition of olive oil or cocoanut oil to the diet significantly altered the rate of solution of the stone. However, this does not show that such vegetable oils might not be of prophylactic value, since the presence of the gall stones in the gall-bladder caused changes in its histology.

4. Biliary stasis if it leads to a definite cholecystitis in the dog definitely delays the solution of the gall stone.

5. Biliary stasis leads to histological changes in the gall-bladder of the dog.

6. The difference in the cholesterin content of dog's bile and human bile per se does not explain why dog's gall-bladder bile dissolves the human gall stone.

7. Soap, especially the soap of lauric acid, is a potent solvent of cholesterin and the mixed type of human gall stone.

8. There is a marked difference between the cholesterin-saponifiable ratio in human bile and dog bile, which we believe explains why gall stones of the cholesterin variety have never been produced in the dog and why the dog's gall-bladder dissolves the human gall stone. We have not proved this, however.

9. The hope of preventing gall stones or of reducing their incidence in pregnancy, or even of dissolving them, is not a "pot of gold at the foot of the rainbow," since it is known that diet influences the cholesterin output in the bile, that the reason why the dog's gall-bladder dissolves the human gall stone is within the realm of the ascertainable, that biliary stasis is preventable, and that the incidence of gall stones in the United States is much higher than in certain Oriental peoples whose diet is quite different from ours.

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Later Results in the Use of Stramonium in Post-Encephalitic Syndrome*†

By A. L. Jacobson, M.D., and F. Epplen, M.D., *Seattle, Washington*

IT IS now a year since we began using stramonium in the treatment of Post Encephalitic sequelae, the first report being read at the 1929 meeting of the American Medical Association in Portland. At that time ninety three per cent of our cases were of the Parkinsonian syndrome, largely because our material came chiefly from the King County Hospital, where a number of these cases had accumulated, and partly because certain cases in that hospital available to us had not been worked over carefully enough to determine that they were post encephalitic in nature, the patients being stowed away in self care wards, pensioners of the hospital. Clearing up the diagnosis on these revealed others of various types.

Price[1] enumerated and classified the various syndromes according to the type or the system involved as follows: (1) motor, (2) sensory, (3) psychic, (4) ocular, (5) aural, (6) respiratory, (7) glandular (hypophyseal), and those of the (8) vegetative system, to which should be added a (9) miscellaneous group. According to Price, only 54% present the Parkinsonian syndrome, 35% some form of excito-motor phenomena, leaving 13% for the miscellaneous group.

The Parkinsonian syndrome is made up of excito-motor phenomena and often shows an admixture of ocular, paretic and phenomena attributable to the vegetative nervous system, e.g. sweating, salivation. Furthermore the Parkinsonian syndrome resembles Parkinson's Disease only in part, the tremor being coarser in the former, the onset and progress differing. Most typical of all differences is the fact that Parkinson's Disease appears late in life, the syndrome at almost any age, our youngest being fourteen. Some of the types, notably the psychic, the glandular and aural types, did not fall into our hands.

Case five of our previous paper belongs to the excito-motor phenomena group. He had a blephorospasm with oculogyric phenomena that partially incapacitated him, and has since then developed a slow, grinding tic of the lower jaw. He reports that this had been present for some six months six years ago and disappeared spontaneously. These movements are slow and occur several times an hour, the

†Presented at the Minneapolis meeting of the American College of Physicians, February 10, 1930.

[1]Unpublished paper read before the King County Medical Society Jan. 6, 1929.

patient is conscious of them but they are beyond his control. They appeared and continue in spite of a regimen of stramonium.

Another patient who is on stramonium is developing a rigidity of the tongue, characterized by him as a fixation, which interferes with mastication and articulation; he also has a typical Parkinsonian syndrome which had very largely yielded to the treatment.

A third patient with a Parkinsonian syndrome, had a habit like tic of the tongue, which consisted of licking the lip and chin every two or three minutes. This symptom was lessened in frequency and degree by stramonium, as did her Parkinsonism.

A fourth patient has a tic of the lips associated with fatigability, both of which were lessened but not eliminated by stramonium.

Of the ocular type, we have seen two cases with oculogyric crises, one upward, the other upward and outward. Both of these cases, more especially the latter, were greatly improved on stramonium.

The respiratory type constitute an interesting group. We have seen only one, a patient on stramonium for post encephalitic fatigability, suddenly, while engaged professionally, developed very deep, slow, slightly irregular dyspnea without cyanosis. This patient had had periodic sighing respiration for about two months preceding. Neither phenomenon has recurred though the patient is no longer taking stramonium.

Vegetative nervous system disorders are usually part and parcel of the Parkinsonian syndrome, notably sweating and salivation, both of which have yielded to stramonium.

Under the miscellaneous group we saw three, two of cerebellar syndrome with ataxia, anergia, dysmetria, incoordination and explosive speech, and one with a group of symptoms almost identical with those of myasthenia gravis: none of these yielded to stramonium.

The ideal cases for medication by stramonium are those of the Parkinsonian syndrome group. Remarkable as it may seem, the worst cases improve the most, and again the very young do exceptionally well. This was well exemplified in a boy of fourteen and a girl of nineteen, both severe cases were rendered almost normal while on large doses, sixty minums four times per day. Both retained a tendency to propulsion and slight tremor, and the boy a slight incoordination, possibly a cerebellar admixture.

Case 1 of our previous series, a patient who was bed-ridden 90% of the time, is now an inmate of the poor farm where it is a prerequisite that they are capable of complete self care. Prior to the use of stramonium he was completely dependent upon attendants. (Moving pictures will be shown of this man.) Case 2, likewise completely dependent upon attendants before taking stramonium, is now doing housework and succeeding in pleasing his mistress. Both these patients from a complete obliviousness to environment, now play cards and checkers. (as will be shown) listen to the radio, attend moving picture theaters, and otherwise demonstrate their interest in life. Case number 6, previously reported, was an automobile mechanic,

disabled to the degree that he could do a few chores about the house, is now able to earn a part of his former salary at his trade.

Our worst case, not previously reported because of space limitations by the editor, bedfast, speechless, with a tremor grotesque in degree, dysphagia limiting his foods to liquids, emaciated, soiling himself constantly because he could not tell of his needs, has been benefitted to the degree that though still bed-ridden, he can speak intelligently to his attendants, is clean, eating and gaining weight. His tremor greatly diminished and his bradykinetic movements so much improved that he can perform certain duties for himself, notably eating.

A woman of 56 with marked tremor, unable to feed herself or walk, very weak and fatigable, the victim of a spinal myalgia requiring morphine, has on stramonium overcome the necessity for opiates, walks and feeds herself and is much less fatigable.

We have now had forty six cases in the hospital and in private practice and have seen twenty others in consultation. Seventy per cent were Parkinsonian syndromes, and only one of which did not get some benefit, to be sure, it was often slight. This was especially true if the patient was young but remarkably great as improvements go in neurology. One patient with a strabismus and one with a hemiparesis were wholly benefitted. So far as these symptoms went these two were more euphoric and had lessened muscular rigidity and were improved in their mental responsiveness. Three cases, of the non-Parkinsonian type of miscellaneous cases. were entirely unbenefitted. This makes about 11% with no benefit. The others were all benefitted from a trifle to a great deal.

Technic

We have been using U. S. P. standardized preparations, specifying a fresh preparation, in doses beginning in adults with twenty measured minums three to four times daily until an optimum dosage is reached, which is usually around sixty measured minums four times daily to get the best results. We have not gone beyond this though it may be necessary on occasion.

In a few of our cases sufficient tolerance has developed so that the dose has had to be increased after a few months. This has been progressive in these cases. Sixty minums have been increased to seventy, then to eighty and then to ninety minims.

Sometimes the relief from tremor in Parkinsonian syndromes is only slight when stramonium is used in spite of striking benefit to the rest of the symptom complex. In these cases it is well to add scopolamine to the medication. We have seen no other indication for the addition of this drug, though Garnett Cheney[2] uses it quite frequently. Our routine, after the optimum dose of stramonium is ascertained and tremor has not satisfactorily subsided, is to add scopolamine 1/150 grain once or twice daily increasing first to 1/120, then to 1/100, later to 1/75 and occasionally to 1/60 grain once or twice daily until the maximum inhibition of tremor is attained or until toxic manifestations preclude large doses. It is striking how after 1/100 grain scopolamine has been added to the morning dose of stramonium it will

[2]Jour. A.M.A. Vol. XCIII, 1929, p. 2030.

eliminate or almost eliminate the troublesome tremor all day. Occasionally the larger doses given twice per day are necessary. Scopolamine, however, does not relax the muscular rigidity and other symptoms nearly as well as does stramonium.

Toxic manifestations from stramonium have been noted. Nausea prevented one case of Parkinsonism from continuing. Mydriasis is always present and troublesome for a time. Contrary to expectations, this usually disappears later in spite of increasing dosage. The ciliary muscle seems to accustom itself to the intoxication remarkably well. Eserine has been recommended, we have found it necessary to counter-act the pupillary dilatation by this drug but once. We used it but once per day in our patient, the young boy previously mentioned and it held the pupils down all day. Several times we have seen cerebral excitation and flightiness with a subjective sensation of nervousness, this necessitated reduction of the dosage. Dowling, of Seattle, reported to us a case of intoxication in a patient who had been on large doses of stramonium without untoward symptoms until she had her prescription filled in a smaller city whereupon she promptly developed symptoms of atropine poisoning. The drug was not analyzed but it is presumed that tr. of belladonna was substituted for the better suited, less toxic, but less used stramonium. Some patients have reported diarrhea but it has not been a troublesome symptom. Marked dryness of the mouth associated with a bitter taste is a constant toxic manifestation, even in patients who had been previously suffering from hyperptyalism.

Summary

1. We still regard stramonium as a valuable drug in palliation of the Parkinsonian syndrome and associated symptoms.

2. Very large doses are necessary.

3. Toxic manifestations are rare and seemingly evanescent.

4. Fresh preparations should be demanded.

5. Particular caution is necessary against substitution of belladonna for stramonium.

6. There are probably at present unknown alkaloids present in stramonium.

Curing the Ulcer Patient*

By SEALE HARRIS, M.D., *Birmingham, Alabama*

CURING the ulcer patient does not consist merely of giving him a routine diet copied from some text book, and prescribing stock prescriptions for alkalies, nor does a gastro-enterostomy, or even resection of the ulcer bearing tissue always result in permanent relief. It is true that many ulcer patients get well and others receive temporary benefit from rest and diet, and surgery relieves many permanently, and others temporarily, but there is still too large a proportion of ulcer patients who receive no permanent benefit from either medical or surgical treatment.

Sir Berkley Moynihan[1] in discussing "the necessity of understanding between the physician and surgeon" says: "The problem of the treatment of duodenal ulcer is one which concerns both the physician and surgeon. A good understanding between them is essential to the welfare of the patient."

"I think it is a reproach to medicine that the surgeon should be compelled to operate so frequently for gastric and duodenal diseases. Such ulcers ought surely to be cured, far more often than they are, by medical treatment."

Eusterman[2] in discussing late recurrences following gastro-enterostomy expressed the opinion shared by most clinicians who see the bad results of the careless, unscientific management of ulcer patients both by physicians and surgeons when he said: "The so-called medical treatment of ulcer has been incomplete, haphazard, and largely aiming at symptomatic relief in 90% of patients;" and he adds that "the complication or sequellae after surgery may be appreciably reduced by proper medical management."

Until recently, we have not had an efficient follow-up system of our own ulcer cases, and we do not know how many recurrences we have had after medical treatment, but they have occurred too frequently. Likewise we have not tabulated the number of recurrences or failures to cure, in ulcer patients who had been treated by others before they came to our Clinic, nor have we yet examined our records for the number of surgical failures that we have been called upon to treat. We are convinced, however, that during the past year we have had more recurring, or uncured, ulcers to treat than ever before; and therefore we have done some serious thinking regarding the causes of medical and surgical failures in the management of ulcer patients.

*Read before the Fourteenth Annual Clinical Session of the American College of Physicians, Minneapolis, Minnesota, February 10-14, 1930.

Early Diagnosis

One of the reasons for the failure to cure gastric and duodenal ulcers, particularly the latter, is that the diagnosis is rarely made by the general practitioner until the lesion has existed for years, when there is organic pyloric stenosis or there are chronic inflammatory changes around the crater of the ulcer that prevent healing.[3]

The old text book triad of ulcer symptoms, i.e., pain, vomiting, and hemorrhage is still the criterion by which many physicians diagnose ulcer. In reality, vomiting and hemorrhage are late symptoms, and usually the ulcer has existed for years before the pain becomes severe enough to impress the patient with the fact that his recurring periods of discomfort, or slight pain two or three hours after eating relieved by taking food, or soda, alternating with months of euphoria, is any more serious than "just a little indigestion."

It is probable that most ulcers of the duodenum, and perhaps some gastric ulcers, if diagnosed early, may be cured by giving the patient frequent feedings of milk, or other bland food, while the patient continues his work; provided that foci of infection, which may be the cause of the ulcer, are removed, and the patient's eating and other living habits are corrected. Usually, however, by the time a patient consults a gastro-enterologist, or a surgeon, the time has passed when the ambulant treatment can accomplish anything, and the patient has to submit to a strict diet that can rarely be carried out, except in a hospital, or he must submit to surgery for relief.

Friedenwald and Finney[4] made a study of 1000 cases of ulcer and the average duration of symptoms of their patients was over ten years. The Mayo statistics[5] showed the average duration of symptoms of their ulcer patients was over nine years. Ulcer patients postpone surgery as long as possible and usually consult the physician earlier, and some years ago a study of several hundred of our cases showed the average duration of symptoms was a little more than five years.[6]

Alvarez[7] in one of the sanest articles that has been written on the treatment of duodenal ulcer says: "Out of 100 patients studied at the Mayo Clinic, in only fifty had changes been made in their diet, and forty-eight had been practically untreated. They had suffered for an average of ten years and had seen an average of four physicians. The diagnosis had been made in thirty-three cases and suspected in twenty-nine more."

"The failure to recognize the disease can be ascribed to the fact that the clinical picture of duodenal ulcer is inadequately described in text books; and the failure to treat ulcer intelligently is due to the deficiencies of text books which describe only one type of management and that one which is not practical for most patients.[11]

Finding and Removing the Cause

Perhaps the reason for some of our failures to cure peptic ulcers is because our attention has been focused too much on the ulcer itself, and we have forgotten sometimes the patient bearing the ulcer; and likewise sometimes we have lost sight of the car-

dinal principle in the treatment of all disease, i.e., find the cause and remove it.

It is true that rarely can we locate with certainty the actual cause of the ulcer in a given case, but if we study the eating and living habits of the patient, and teach him the personal hygiene necessary to build up his resistance to infections and remove the foci of infections that exist when treatment is begun, we have contributed much to his chances for cure whether treated medically or surgically.

Medical literature is plethoric with discussions of various theories on the etiology of peptic ulcer, though very little has been proved that will account for the apparent increase in the number of cases. The work of Rosenow[8] which has been verified by a number of other research workers[9] seems to make it certain that infection is present in all cases and that the streptococcus viridans, or other pathogenic organism is the probable exciting cause of gastric and duodenal ulcers. It likewise seems certain that there is an underlying local or constitutional condition that lowers the resistance of the individual to the infections that produce ulcer.

Dantzer[10] believes that we should regard ulcer as a constitutional condition, with an abnormality of the circulation in the mucosa of the stomach as the predisposing cause. Many others hold to the theory of vascular changes in the ulcer bearing area as being the essential factor in the pathogenesis of ulcer; but what is the cause of the circulatory pathology, both local and constitutional?

Vitamin Deficient Diets

It certainly is a fact that the eating habits of most ulcer patients need to be corrected; and some of the facts brought out in recent studies of nutrition, particularly with reference to diets deficient in vitamins seem to indicate that one of the important predisposing causes of ulcer is the general use of devitaminized foods.[11]

About fifteen years ago, McCollum, Simons and Parsons[12] expressed the opinion that "the rôle of food in the etiology of many diseases involves increased susceptibility to infection due to lowered resistance caused by faulty diet." Others interested in nutrition have come to the same conclusion, but the work of McCarrison[13] seems to furnish some proof that foods of low vitamin content if used over long periods of time predispose to ulcers and other infections of the gastro-intestinal tract.

The great fault of the American diet is the excessive use of carbohydrates. Sugar saturated, vitamin starved Americans, i.e., those who live largely on white flour bread; white potatoes; white rice; white sugar, with which they saturate their coffee, soft drinks and desserts; lean meats; and oleomargarine butter; are prone to ulcer. Therefore, we do not get very far in curing ulcers by feeding the patient devitaminized diets; and we may expect recurrences, if after the patient has had a medical "cure" or a gastro-enterostomy, or resection of the ulcer, he is allowed to go back to the same unbalanced, deficient vitamin diet he was eating when he developed the ulcer.

We have constructed a diet based upon our modification of the original Lenharz diet[14] which is properly balanced and which meets the vitamin requirements of the average malnourished adult ulcer patient. No one diet can be prepared which will suit the needs of every ulcer patient, but this diet, while keeping the vitamin content high, may be added to, or subtracted from, to meet the nutritional requirements of the under-weight or over-weight ulcer patient.

The Relation of Over-Work to Ulcer

Improper food is surely not the only predisposing cause of ulcer, and a proper diet is not the only consideration in curing the patient. More than twenty years ago, Kauffman[15] called attention to the fact that patients who were in a lowered state of vitality from chronic fatigue were prone to ulcer and likewise were more susceptible to recurrences after treatment. We are convinced that Kauffman is correct in his idea that over-work is a predisposing cause of ulcer. We have had too many ulcer patients who had recurrences both after medical and surgical treatment who gave a history of overwork and long hours, not to have rather strong convictions that rest and prolonged rest, is an important factor in curing the patient; and that if the patient would not have recurrences after treatment, he must regulate his life so that he can have sufficient rest to keep in good physical condition.

Rest bears much the same relationship to the treatment of ulcer that it does in the cure or arrest of tuberculosis and other chronic diseases in which there is local pathology engrafted on a constitutional condition.

We have noted also that the neurasthenic and psychasthenic element is present in many ulcer cases; and surely the best method of treating psycho-neurasthenia is the Weir-Mitchell rest cure.

Rest In Bed Necessary

Moynihan[16] asserts that it takes a long time for the gastric or duodenal ulcer to heal. Therefore, he insists that several months in bed is sometimes essential in the medical cure of ulcer; and he believes that fewer cases will come to surgery, if they will take a sufficiently long rest in bed while at the same time receiving the proper diet and medical treatment.

Two months in bed is considered best by many, but we have found that from three to four weeks in bed, with the avoidence of overwork afterwards is usually sufficient to cure the ulcer that can be relieved medically.

The fact that the patient is free from pain does not mean that the ulcer has healed because every surgeon who has had much experience with ulcer knows that when the patient is operated upon in the quiescent stage the ulcer often shows no signs of healing; and many autopsies on persons who never in their lives had ulcer symptoms and who died of other causes, show active ulcers that must have existed for many years. Therefore, the fact that the ulcer patient becomes free from pain in a few days after beginning the diet does not mean that he should not rest in bed for several weeks while undergoing thorough treatment.

Abstinence From Tobacco Important in Curing the Ulcer Patient.

Much has been written by the Germans and the English regarding the use of tobacco as a predisposing cause of ulcer of the stomach and duodenum. Most of the gastro-enterologists of the United States are convinced of the relationship of tobacco to ulcer, though some, notably those who smoke excessively themselves do not believe that tobacco injures anyone in any way at any time.

Sir Berkeley Moynihan[17] recognized as one of the greatest authorities on ulcer in the world does not equivocate in expressing his opinion regarding the influence of tobacco in the pathogenesis of ulcer. In a recent address he said:

"Among the most harmful of habits for all these patients is smoking. We have found that in many cases of jejunal ulcer a hyperchlorhydria is present and may be extreme. If a Rehfuss meal is given to a patient accustomed to tobacco at a time when he is not smoking, his normal 'curve' may be recorded; if a second meal is given while a pipe is being smoked, the increase in gastric acidity is very striking. In some cases the excess of free HCL may be slight, but its secretion continues over a long period; in a few cases these two effects of tobacco, increased secretion and increased duration of secretion, are combined. An 'attack' of duodenal ulcer often follows an orgy of tobacco; and many attacks are checked by abstinence from it. 'Attacks' ascribed to duodenal ulcer are sometimes due only to nicotine poisoning, and I have not seldom rescued patients from impending operations by noticing their deeply stained fingers and by prescribing for them a respite from tobacco for a few months and a diminished indulgence in it forever. The close mimicry of hunger-pain in nicotine intoxication appears to have escaped notice."

Moynihan presents charts[18] to illustrate the effects of tobacco on the free acid in the gastric juice. In each test these show a marked increase after smoking.

Tyrrell Gray[19] another British surgeon in discussing tobacco as a predisposing cause of ulcer said:

"The predisposing factor in duodenal ulcer is the relative increase of vagus excitation established by diminution of sympathetic control, or by increasing vagus stimulation. There is an outstanding example of an alkaloid which paralyzes sympathetic ganglion cells—that is nicotine. On these grounds I have for some years prohibited smoking in duodenal ulcer."

"The proportion of men to women who have ulcer is roughly four or five to one (in itself perhaps some indication of the influence of nicotine) and 96 percent of the males were smokers. Of the smokers, 22 percent abandoned the habit, or nearly so, with the result that 90 percent were cured, 8 percent greatly relieved, and 2 percent only failed. Where tobacco was unchecked, only 47 percent were cured, and 12 percent recurred. Recurrence is four times as frequent in those who continued smoking."

Eusterman[20] of the Mayo Clinic, n discussing the unfavorable effect of tobacco on ulcer patients says:

"The excessive use of tobacco is deleterious to the health of the patient with peptic ulcer. In those susceptible to the influence of nicotine, moderate amounts may be harmful. The patient who craves tobacco invariably consumes excessive amounts and the habit should be discouraged. Langley showed that nicotine paralyzes the synapses of the sympathetic nervous system, so that dyspeptic symptoms in habitual smokers are logical, owing to the unopposed vagal action. Wagner concluded from a recent investigation that all the subjective and roentgenologic signs of duodenal ulcer can be produced by the excessive use of tobacco. During the last decade the typical syndrome of peptic ulcer has been occasionally observed in young

adults given to excessive cigarette smoking, and their discomforts have disappeared largely through the discontinuance of the habit. German clinicians are loath, or refuse, to accept for treatment the patient with peptic ulcer whose fingers are tobacco-stained. I have frequently noticed the peculiar psychologic fact that patients of physicians who are inveterate smokers are not, as a rule, warned to discontinue or restrict the use of tobacco."

"The definitely better end-results that are obtained in either the surgical or non-surgical treatment of ulcer in women should furnish a therapeutic hint and justification for postoperative precautions. While factors of an anatomic, physiologic, and occupational nature may play a part, I feel that such greater success is due more to their wholehearted and continued co-operation regarding matters of diet and mode of eating, and to the fact that generally speaking they are not handicapped by such bad personal habits as the excessive use of tobacco and alcohol.

If tobacco is a predisposing cause of ulcer, it would seem that since smoking among women is becoming almost universal, that one of the by-products of "feminine freedom" will be an increase in the incidence of ulcer among them. It happens that the only case of gastro-jejunal ulcer in a woman that we had last year was that of a movie actress who smoked cigarettes excessively. She had been operated upon six months before she came to us by a surgeon of national reputation and there could not have been any faulty technic in the operation. It was not until she reduced her tobacco that she improved. She never could be induced to give up smoking entirely, and she probably will have a recurrence of her ulcer.

It is difficult to prove that tobacco is a predisposing cause of ulcer, just as it is difficult to prove that an alcoholic debauch is responsible for some of the perforations in ulcer; yet I and others have observed a number of cases of perforations in ulcer patients after drinking excessively. Still on the authority of such men as Sir Berkely Moynihan, Sir Humphrey Rolleston, Dr. Tyrrell Gray, Eusterman and others, it would seem wise to instruct our ulcer patients to discontinue the use of tobacco for the rest of their lives.

Curing the Surgical Ulcer Patient

The mortality following ulcer operations can be reduced and many recurrences prevented by the proper preparation of the patient for the operation and by careful post-operation medical supervision. Marginal or jejunal ulcers, likewise may be prevented if the patient is given the proper instructions regarding diet and hygienic living after operations.

One of the attractions that surgery offered in the past to the ulcer patient was the then current belief of the average layman that after operation for ulcer he could do what he had always been doing and eat what he wants. Many patients who have had recurrences or marginal or jejunal ulcers, or other complications following gastro-enterostomy have become disillusioned about surgery curing ulcer of the stomach in every case, until now many people have a holy dread of an operation for ulcer.

The surgeon themselves are partly to blame for this condition because they often quote a distinguished surgeon who is said to have remarked: "If a man can't eat what he wants after a gastro-enterostomy there is no

use having it done." Probably what he meant was that a man after a gastro-enterostomy can eat all the food he needs, because it would seem that a surgeon would know that a gastro-enterostomy does not remove the cause of an ulcer of the stomach or duodenum; and if the patient in a few weeks after operation goes back to the same eating habits, or the same manner of living which brought on the ulcer, he runs the risk of recurrence of the ulcer, or of the development of a marginal or jejunal ulcer after operation.

The practice of surgeons in getting an ulcer patient up two weeks after operation probably accounts for some of the failures in ulcer surgery. This is particularly true after a gastro-enterostomy, in which the ulcer is not removed. The duodenal ulcer is placed in a favorable condition for healing by a gastro-enterostomy, but an ulcer that has existed for years cannot be expected to heal in two weeks, or even three weeks. Therefore three weeks, and in some cases four weeks, in bed following a gastro-enterostomy will give the ulcer patient a better chance for permanent cure than if he is hustled out of the hospital two weeks after his operation. Healing even after clean surgery cannot be said to be complete in two weeks, and at least three weeks rest in bed following ulcer surgery of any kind without doubt is best for the patient.

Amateur Gastric Surgery

An important consideration in curing the surgical ulcer patient is the selection of the surgeon, because the immediate mortality and the post operative complications of gastric operations at the hands of amateur surgeons is something frightful. It is perfectly true that many surgeons operating in small hospitals are doing first-class abdominal surgery, but it also is a fact that with the multiplying community hospitals many general practitioners are performing, or rather are attempting to perform, gastro-enterostomies and their faithful patrons are the sufferers from their lack of preparation for doing major surgery.

I have not seen any statistics of the results following gastro-enterostomies by general practitioners who do one or two gastro-enterostomies a year; but the study by Verbryck[21] of the end results in gall-bladder surgery in a Washington hospital with an open staff, shows the tragedy of general practitioners attempting to do surgery for which they are not prepared. Verbryck showed that in a splendidly equipped Washington hospital that the mortality of one surgeon who performed 62 gall-bladder operations a year was 1.6 per cent; while the percentage of those who died at the hands of the physicians performing less than ten gall-bladder operations a year averaged 14 per cent. In one case of the occasional operator the mortality was 28 per cent.

Eusterman[22] in a paper published in 1919 mentions 300 gastro-enterostomies performed by many surgeons in various parts of the country which had to be undone in the Mayo Clinic; and in a study of 84 of their gastro-jejunal ulcers, concludes that they occur in 1.5 to 2 percent of gastro-enterostomies. It perhaps would discourage

the amateur surgeon if it were possible to obtain, and publish, the number of gastro-enterostomy patients who had to be re-operated upon in the United States every year.

The physician must let his conscience be his guide as to whether or not he is adequately prepared and the hospital in which he operates properly equipped for gastric surgery, because the lives and future health and happiness of his trusting patients are in his hands.

Dietary Management Before and After Ulcer Operations

The dietary management of the ulcer patient before operation should be entirely different from that employed in the medical treatment; because the most important problem with the operative ulcer case is to prevent acidosis; and some of the stock diets as advised for the medical treatment of ulcer if used for a few days before operation will surely result in the retention in the body of incompletely metabolized fatty acids, the essential factor in acidosis. It, therefore, is inadvisable to give milk, cream, butter, or other fats to an ulcer patient within 24 hours of operation, but carbohydrates should be given freely, preferably strained orange juice and ten percent solution of dextrose, corn syrup, or honey.

The time required to properly prepare an ulcer patient for operation varies greatly, and depends to a great extent upon his state of nutrition. The well nourished ulcer patient may be prepared for operation in one or two days, but the emaciated, anemic, debilitated ulcer patient, who is dehydrated and has no vitality will stand a better chance for recovery after operation if he can be placed in a better state of nutrition. Certainly no patient with gastric retention should be operated upon without a thorough study of kidney function. If the blood urea, or the non-protein nitrogen is high, the patient should be given only liquids, largely soluble carbohydrates until the kidneys are functioning normally.

Sir Berkley Moynihan stresses particularly the preparation of ulcer patients for operation, stating that it often requires weeks to get them prepared.

The dietary after care of ulcer operative cases is also most important. No fats should be given in three or four days after operation and not then if the patient is vomiting or shows other symptoms of acidosis. Likewise the stock diets have no place in the dietary management of ulcer patients within two weeks after operation.

The prevailing custom among surgeons of allowing ulcer patients to go on a general diet, "eat what you want," two weeks after a gastro-enterostomy, or other operation for ulcer, is responsible for the failure to cure many patients. On questioning most of the ulcer patients who were not relieved by surgery, or who had recurrences, or marginal ulcers, and who came to us for medical treatment, they almost invariably said that their surgeons had told them they could "eat what they wanted two weeks after operation," and few of them remembered having been given any instructions whatever regarding diet, or personal

hygiene, during the time of their convalescence after their operations. It may be added that a number of these patients had been operated upon by surgeons of great reputation.

When surgeons consider the pathology of gastric or duodenal ulcers, they must know that healing cannot take place in two weeks. They also should realize that there is more to surgery than the mere mechanics of an operation; that the surgeon's obligation to his patient does not end when the wound he has made has healed. Certainly surgeons will have better results following gastric surgery if they are more careful in dieting their patients after operation. They should inform their patients that it takes an ulcer a long time to heal after a gastro-enterostomy, and that the original cause of the ulcer has not been removed by operation; and that if he would not have a recurrence he must be careful about his diet for six months or a year after he leaves the hospital. What is still better, surgeons, when possible, should have the cooperation of physicians of ample experience in the dietary management of their ulcer patients. They should also follow up such patients for from six months to a year to see that they carry out the proper diet.

System of Hygienic Living for Ulcer Patients.

The great majority of properly treated ulcer patients, whether medical or surgical, may look forward to becoming more efficient and to enjoying life more than they ever did before; provided they follow the same rules of personal hygiene that every man in health should observe. The ulcer patient perhaps will live longer than he would have had he not developed an ulcer, because if taught properly how to live, as the physician or surgeon has the opportunity of doing, he will take such good care of himself that he will be less susceptible to many of the infirmities of middle life, and old age will thus be deferred.

A copy of the following outline of a system of hygienic living with copies of simplified diet lists prepared to suit his particular needs is given to each of our ulcer patients before they are dismissed.

1. THE PROPER DIET—Three medium meals a day at the same hours, and a glass of milk between meals for at least three months after treatment; then one raw fruit, one raw vegetable, two cooked tender green vegetables, a pint to a quart of milk every day for the rest of his life. Meat and light desserts, not more than once a day. Enough bread, potatoes, rice, butter and other simple foods to maintain the normal weight. Thorough mastication of food is most important.

2. WORK—AVOID OVERWORK—Six to eight hours of honest work a day for five days a week and three or four hours on Saturday.

3. SLEEP—Eight or nine hours in bed at night; and lie down, sleep, if possible, for half an hour after the noon meal.

4. EXERCISE—Fifteen minutes room exercises with windows wide open, before the morning bath. Follow the bath with massage (brisk rubbing with open hand) of the entire body until the skin is reddened. A

walk of one or two miles in the open air and sunlight each day, or what is better, play golf two or three afternoons a week.

5. RECREATION—Eight hours for play includes morning exercises, bath, golf, or other outdoor exercises; time for eating at least ½ hour for each meal, perhaps an hour for dinner and no work or serious reading after six o'clock in the afternoon. Frequent week-end trips and an annual vacation of from two weeks to a month every year.

6. SERENITY — Worry, anger, grief, abnormal fears, or other emotional disturbances will break down resistance to infections. Therefore, the cured ulcer patient should cultivate serenity in all the relations of life.

7. ELIMINATE THE TOXINS —Coffee, tea and the so-called Cola drinks, which contain caffeine, a habit forming drug, should not be used by the ulcer patient, except occasionally as a stimulant after losing sleep; or as a drug for headache or shock. Tobacco and alcohol are predisposing causes of ulcer and are quite injurious to the ulcer patient. Therefore, he should never use them in any quantity, even after his ulcer has been pronounced cured.

8. THE ANNUAL OR SEMI-ANNUAL PHYSICAL EXAMINATION—Every adult should have an annual, or semi-annual, physical examination even when in health. The ulcer patient should report to his physician once a month for six months, and then for the rest of his life have an annual or semi-annual physical examination, with particular reference to a possible return of the ulcer; or the possible development of a cancer of the stomach, which, if diagnosed early, can be cured by operation.

Each section of the above outline of hygienic living is discussed with the patient and he is shown how he can conform his daily habits to a regular systematic regimen. He is also impressed with the fact that his future health, efficiency and happiness depend largely upon his living the simple hygienic life, that he must do his part in preventing a recurrence of his ulcer.

TABLE 1. ULCER DIET FOR THE FIRST WEEK OF TREATMENT.

Amount			FOOD
Gm.	Oz.	Table-spoonsful	
			First Day
15	½	1	Every hour from 7 a.m. to 7 p.m., ½ ounce of a mixture of 2½ ounces of cream to 4 ounces of milk (thirteen feedings)
15	½	1	Strained orange juice after the milk and cream at 7 a.m., 1 p.m. and 7 p.m.
			Second Day
30	1	2	Every hour from 7 a.m. to 7 p.m., 1 ounce of a mixture of 4 ounces of cream and 9 ounces of milk (thirteen feedings)
30	1	2	Strained orange juice after the milk and cream at 7 a.m., 1 p.m. and 7 p.m.
			Third Day
45	1½	3	Every hour from 7 a.m. to 7 p.m. (inclusive), 1½ ounces of a mixture of 7½ ounces of cream and 12 ounces of milk (thirteen feedings)
45	1½	3	Strained orange juice after the milk and cream at 7 a.m., 1 p.m. and 7 p.m.
			Fourth Day
60	2	4	Every hour from 7 a.m. to 7 p.m. (inclusive), 2 ounces of a mixture of 8 ounces of cream and 18 ounces of milk (thirteen feedings)
60	2	4	Strained orange juice after the milk and cream at 7 a.m., 1 p.m. and 7 p.m.
			Fifth Day
75	2½	5	Every hour from 7 a.m. to 7 p.m. (inclusive), 2½ ounces of a mixture of 12½ ounces of cream and 20 ounces of milk (thirteen feedings)
60	2	4	Strained orange after the 7 a.m., 1 p.m. and 7 p.m. feedings of milk and cream.
			Sixth Day
90	3	6	Every hour from 7 a.m. to 7 p.m. (inclusive), 3 ounces of a mixture of 15 ounces of cream and 24 ounces of milk (thirteen feedings)
60	2	4	Strained orange after the 7 a.m., 1 p.m. and 7 p.m. feedings of milk and cream mixture.

TABLE 2. ULCER DIET FOR THE SECOND WEEK OF TREATMENT.

Amount			*Seventh to Tenth Days.*
Gm.	Oz.	Tablespoonfuls	8 A.M. BREAKFAST:
90	3	6	Strained orange juice
90	3	6	Strained oatmeal or one shredded wheat biscuit, toasted, or 1 slice dry toast of whole wheat bread
90	3	6	Milk
			One soft boiled egg
10	1	1	Pat butter
60	2	4	Cream
			10 AND 11 A.M.:
90	3	6	1 ounce of cream and 2 ounces of milk
			1 P.M. DINNER:
90	3	6	Strained tomato juice
25	1	1 large	Scraped beef, lightly broiled
90	3		Milk
30		(1 slice)	Whole wheat bread, toasted
			3 AND 5 P.M.
90	3	6	1 ounce of cream and 1 ounce of milk
			6 P.M., SUPPER:
90	3	6	Strained orange juice
90	3	6	Strained oatmeal or one shredded wheat biscuit, toasted, 1 thin slive dry toast of whole wheat bread
20			
60	2	4	Cream
			One soft boiled egg
			One pat butter
90	3	6	Milk
			9 P.M.
90	3	6	2 ounces of milk and one ounce of cream.

TABLE 3. ULCER DIET FOR THE ELEVENTH TO FOURTEENTH DAYS.

Gm.	Oz.	*Amount* Tablespoonsful	
			8 A.M., BREAKFAST:
90	3	6	Orange juice (strained)
90	3	6	Strained oatmeal or one shredded wheat biscuit
90	3	6	Cream
			One soft boiled egg
30		(1 slice)	Whole wheat bread, toasted
10		(1 pat)	Butter
			10 A.M.:
120	4	8	Milk
			11 A.M.:
120	4	8	Milk
			1 P.M., DINNER:
90	3	3	Strained tomato juice
50	2	2 large	Scraped beef or minced breast of chicken
30		(1 slice)	Dry toast, whole wheat
10		(1 pat)	Butter
60	2	2	Ice cream
			3 P.M.:
120	4	8	Milk
			5 P.M.:
120	4	8	Milk
			6 P.M., SUPPER:
90	3	6	Orange juice, (strained)
			One soft boiled egg
30		(1 slice)	Whole wheat bread, toasted
10		(1 pat)	Butter
			9 P.M.:
120	4		Milk

TABLE 4. ULCER DIET FOR THIRD WEEK OF TREATMENT.

Amount Gm.	Oz.	Tablespoonsful		*Fifteenth Day to Twenty-first Day*
				8 A.M., BREAKFAST:
90	3	6		Strained orange juice or strained grapefruit juice
90	3	6		Strained oatmeal
90	3	6		Cream
				Two soft boiled eggs
30		(1 slice)		Toast, whole wheat bread
10		(1 pat)		Butter
150	5	10		Glass of milk
				11 A.M.:
150	5	10		Glass of milk
				1 P.M., DINNER:
120	4	8		Strained tomato juice or strained vegetable soup
100	4	4	large	Scraped beef or minced breast of chicken
100		4		Tender green vegetables, as turnip greens, spinach or string beans (mashed through sieve)
30		(1 slice)		Toast, whole wheat bread
20		(2 pats)		Butter
120	4	4		Ice cream, cup custard, boiled custard or gelatin and cream
				4 P.M.:
120	5	10		Glass of milk
				6 P.M., SUPPER:
120		8		Thick puree of peas or beans
100		4		Tender green vegetables, as turnip greens, spinach or string beans mashed through sieve
30		(1 slice)		Toast, whole wheat bread
20		(2 pats)		Butter
150	5	10		Glass of milk
90	3			Strained orange juice
				9 P.M.:
150	5	10		Glass of milk

TABLE 5. ULCER DIET FOR FOURTH, FIFTH AND SIXTH WEEKS OF TREATMENT.

Gm.	Oz.	*Amount* spoonfuls Table-	*Fourth to Sixth Weeks*
			8 A.M., BREAKFAST:
90	3	6	Strained orange juice
90	3	6	Strained oatmeal, or ½ shredded wheat biscuit
90	3	6	Cream
			One egg, soft boiled, poached or scrambled
60		(1 slice)	Dry toast, whole wheat bread
20		(2 pats)	Butter
			10 A.M.:
150	5	10	Milk
			1 P.M., DINNER:
120	4	8	Strained tomato juice, clear broth or tomato broth, or strained vegetable soup
100	4	4 large	Scraped beef or minced chicken or lamb
100		4	Turnip greens, spinach or string beans, mashed through a sieve
60		(1 slice)	Dry toast, whole wheat bread
20		(2 pats)	Butter
120	4	4	Ice cream, boiled custard or gelatin, or watermelon or cantaloup juice
			4 P.M.:
150	5	10	Glass of milk
			6 P.M., SUPPER:
120	4		Thick puree of peas or beans
90		3	Turnip greens, spinach or string beans, mashed through a sieve
60		(1 slice)	Dry toast, whole wheat bread
20		(2 pats)	Butter
120	4	8	Milk
120	4	8	Strained orange juice
			9 P.M.:
150	5	10	Glass of milk

TABLE 6. ULCER DIET AFTER SIX WEEKS.

BREAKFAST:

One fruit::Strained orange or grapefruit juice.

One Cereal: Small portion of thoroughly cooked oatmeal, or 1 shredded wheat biscuit with ½ glass cream—no sugar.

Eggs: 1 or 2 eggs poached or soft boiled, or soft scrambled.

Bread: 1 slice whole wheat bread, 2 or 3 pats butter, 2 tablespoonful honey.

Milk: 1 glass sweet milk.

Three hours after breakfast take glass milk (one-third cream)

DINNER:

Soup: Strained chicken, celery, vegetable, barley soup, or strained tomato juice.

Tender Meats: (Small portion) Broiled, boiled, or baked—not fried Small portion of chicken, turkey, mutton, roast beef, bacon, thinly sliced boiled ham, or fish.

Tender green vegetables: (one or two varieties) Large serving spinach, turnip greens, tender beans, cooked without much grease. Butter or mayonnaise or olive oil and lemon juice may be used freely on vegetables after they have been cooked.

Bread: One slice whole wheat bread or dry toast or small piece of country ground corn meal bread, or 1 small corn muffin; two or three pats of butter.

Milk: 1 glass sweet milk, or buttermilk, (one third cream).

Dessert: Soft part of baked apple, thoroughly ripe banana, or other fruit with cream—no sugar. Ice cream, or gelatin, or sherbet twice a week.

Three hours after dinner take glass of milk (one-third cream.)

SUPPER:

Soup: Puree (thick strained soup) of peas or beans, or oyster stew—no oysters.

Tender green vegetables: Large serving of spinach, turnip greens, mustard greens, tender string beans, cooked without much grease.

Bread: 1 slice whole wheat bread or dry toast. 2 or 3 pats butter.

Milk: 1 glass sweet milk, or buttermilk (one third cream)

Dessert: Strained orange juice, soft part baked apple, or very ripe banana and cream without sugar, or gelatin, or egg custard.

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Unusual Addison's Syndromes*†

By A. B. Brower, M.D., F.A.C.P., *Dayton, Ohio*

"THE leading and characteristic features of the morbid state to which I would direct attention are anemia, general languor and debility, remarkable feebleness of heart action, irritability of the stomach, and a peculiar change in color of the skin occurring in connection with diseased condition of the suprarenal capsule." This original description of adrenal insufficiency by Addison[1] remains classical.

If a patient presents these clinical phenomena and at autopsy shows bilateral destructive changes of the adrenals, there can be no doubt as to the diagnosis. On the other hand, if a patient presents the clinical manifestations of Addison's disease, and at autopsy no abnormalities of the adrenals are revealed, it is usually assumed that there has been destruction and impairment of function of the chromaffine system outside of the adrenals. Addison described a clinical and not a pathological concept.

What are the functions of the adrenal glands? The gland is composed of two distinct layers—the medulla and cortex. The medullary portion is composed largely of chromophil cells of central nervous system origin. This portion is not essential to life, but is the source of the only known active principle—epinephrin. It is generally assumed that the hormone enters the circulation by way of the central veins of the gland. This secretion has a pronounced effect on the functions of structures innervated by the sympathetic nervous system. The cortex is composed of epithelial areas derived from the wolffian body. This portion is essential to life. It is generally accepted that the cortex furnishes a substance affecting growth and reproduction; attempts to isolate the vital hormone have not met with success. It might be well to mention that the complete Addison's syndrome has never been produced experimentally.

The pathological conditions of the adrenals most often found at autopsies performed on patients showing the typical Addison's syndrome are tuberculosis, syphilis and malignancy; in rare instances, pressure atrophy, lymphadenoma, mycosis fungoides, infarcts in children, roentgen ray necrosis, or hypoplasia is encountered.

In a recent survey made by Barker[2] of the Mayo Clinic, of twenty-eight cases of Addison's disease which came to autopsy, the following was found: In twenty-five of the cases advanced bilateral tuberculosis of adrenals was

*From the Diagnostic Division of the Dayton Clinic, Dayton, Ohio.

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demonstrated; in three, bilateral atrophy was found; in eleven of fourteen cases autopsied in the last four years, an acid-fast bacillus resembling the tubercle bacillus was found in the sections of the gland. In twenty-five cases, healed tuberculosis was found in the lungs. In thirteen cases, tuberculosis was active in the lungs. In six cases, active tuberculosis was found in the genito-urinary organs, while in three instances, active tuberculosis was found in the lymph glands, spleen, and liver. In only three cases was there no evidence of active tuberculosis in the adrenals. In this series, there was one case of syphilis of the adrenals. That syphilitic adrenalitis may be a more frequent etiological factor in the development of Addison's disease than is generally believed is indicated by the recent studies of Warthin.[3] The writer has observed the development of many of the symptoms of Addison's disease after influenza infections and after severe stress and strain, as was experienced during the World War; in these cases these symptoms were transitory.

The cause of the pigmentation in Addison's disease has given rise to much investigation and conjecture. According to Loeper[4] the etiological factors fall into four groups: adrenal origin, cachectic, nervous, and mixed origin. Most investigators believe that both the adrenal glands and the sympathetic nervous system are factors. Lowered suprarenal secretion lessens the ability of the tissues to eliminate sulphur. The high sulphur content of the blood in the presence of melanoderma suggests that this is an important factor in the production of excessive melanin pigmentation. Bittorf[5] suggests that the increase of oxidase in the skin plays an important rôle.

Addison's disease manifests itself in four distinct forms; gastro-intestinal, painful, melanodermic, and asthenic. In the painful and melanodermic types, the involvement of the sympathetic system appears to dominate the clinical picture. In the asthenic type, the adrenal insufficiency is most pronounced. In analysing such a classification we discover the outstanding symptoms as described by Addison. The four most common clinical characteristics are asthenia, pigmentation, gastro-intestinal disturbance, and hypotension. At least three of these symptoms are necessary to make a diagnosis. Anorexia, nausea, vomiting, gaseous eructation, and meteorism are common complaints. Alternating attacks of constipation and diarrhea are frequent symptoms. Loss of weight is quite constant. Patients often experience lumbar and abdominal pains, and, in some cases, anginal pains occur, despite the presence of hypotension. Dyspnea is more likely to be a terminal condition and is frequently associated with syncope and collapse, due to feeble heart action and extreme terminal hypotension. The heart sounds are weak. The presence of hypotension is not essential to a diagnosis of Addison's disease, since a systolic pressure of 120 or above has been reported in a number of cases. However, the majority of patients have a systolic blood pressure below 100. A study of the literature reveals that pigmentation is rarely absent. In these cases in which melanoderma is absent, it is difficult to establish a diagnosis of

Addison's disease. Melanosis usually occurs more distinctly on exposed areas and on approximating surfaces. All mucous membranes are usually pigmented, especially the lips.

The laboratory investigations in this disease have yielded important data. Contrary to Addison's statement, anemia is not a cardinal feature. The vast majority of cases show a marked lymphocytosis. Blood sugar values usually fall within normal limits. According to Brown,[6] of the Mayo Clinic, the circulating blood and plasma were normal in all cases. Electrocardiographic tracings show no abnormalities characteristic of this disease. Urine analysis usually shows a normal output with a slight amount of albumin and a few hyaline and finely granular casts. Some observers have found that as many as 40 per cent of their patients with Addison's disease showed glycosuria. This has not been our experience, nor have the investigators at the Mayo Clinic found this to be true. Lowered renal function seems to be the rule, which is rather to be expected in view of the marked circulatory asthenia. No appreciable disturbance of hepatic function has been demonstrated. Lowered gastric acidity or complete achlorhydria is the rule. A lowered basal metabolism rate was found in thirty-three per cent of the cases in which metabolic determinations were made.

In the differential diagnosis of Addison's disease, those conditions in which diffuse cutaneous pigmentation is a cardinal sign should be carefully considered. In pernicious anemia, the skin has a lemon tint as opposed to the brownish discoloration in Addison's disease; the blood picture will aid in the solution of this problem. In malignancy, we find more wasting and the discovery of the neoplasm will clear the diagnosis. Arsenic poisoning will frequently give a similar skin color; here we find the history, lack of mucous membrane pigmentation, and the presence of hyperkeratosis helpful in establishing the diagnosis. The Marsh test of the urine, before and after giving sodium thiosulphate, will often be of value in such cases. Pregnancy occasionally causes marked increase in pigmentation, but in these cases the muscular and vascular asthenia is not so great; the history and vaginal examination will give the correct diagnosis. Various liver conditions give rise to color changes in the skin; the change in the size of the liver, the icterus index, hepatic function tests, the presence or absence of bilirubinuria, and acholic stools will aid in the differtiation. Hemachromatosis gives a distinct pigmentation; the presence of hypertrophic cirrhosis, glycosuria, and histologic examination of the skin for hemosiderin deposits will establish this diagnosis. Malarial pigmentation does not affect the mucous membranes and the finding of the plasmodia makes this diagnosis definite. Patients with pulmonary tuberculosis occasionally will show skin pigmentation, but the chest findings and the degree of asthenia are of value in differentiating this condition from Addison's disease. It must be borne in mind, however, that active pulmonary tuberculosis and Addison's disease will be often found in the same patient. The history of the case and the bluish-gray pigmentation are the important

considerations in argyria. Dementia, delirium, diarrhea, and the restriction of pigmentation to exposed areas are the cardinal signs and symptoms in pellagra. The pigmentation occasionally associated with Graves' disease is to be distinguished by hypertension, rapid and strong heart action, palpable thyroid, and the increased metabolic rate. The occasional pigmentation in lues can be identified by the history, lack of hypotension and asthenia, and by the Wassermann and Kahn reactions. The degree of fatigability of a muscle can be studied by ergography and is more pronounced in Addison's disease.

Two cases have been encountered recently, which, although presenting the typical clinical symptoms described by Addison, showed at autopsy pathological conditions which are very unusual.

Case 1. A single girl, twenty-eight years old, was referred to us by a surgeon whom she consulted relative to the advisability of having her thyroid gland removed. She complained of asthenia, pigmentation, rapid heart, nervousness, and pains through her chest and back. Her mother was living but had pernicious anemia. Her father was living and well. She had four brothers and three sisters all in good health. Her babyhood, childhood, and adolescence were uneventful. The only acute infectious disease was influenza during 1917. There was no history of venereal disease, surgical operations, or serious injuries. She never used tea, coffee, tobacco, or alcohol.

Her maximum weight was 145 pounds; the present weight was 109 pounds. The patient said she had not been well for three years. She had been treated for bronchitis and liver trouble, and for the past year had been under treatment for goiter. During this time she had gradually grown worse. Since the onset of illness she experienced the following symptoms: many headaches, far-sightedness, frequent colds with cough, some enlargement of the thyroid gland, and indigestion. During the development of these symptoms, she observed a change in the color of her skin, but thought and was told that she was jaundiced. She experienced constipation and generalized pain involving the joints, back and chest. For the past year she had a rapid heart, nervousness, and some loss of weight.

Physical examination showed a tall, thin girl, with a yellowish-brown skin. The buccal and vaginal mucous membranes showed a similar brownish pigmentation. The temperature was 97, pulse 84, respiration 18. The pupils reacted to light and in accommodation. The retinae showed no pigmentation. The ears and nose were negative. The tonsils were moderately infected. The teeth showed moderate pyorrhea. Moderate general lymphadenopathy was found. The thyroid was palpable, soft, moderately enlarged, and somewhat tender. The breasts were negative. The heart showed no deviation from the normal. A few râles were heard over the right hilus. The liver, spleen and kidneys were normal in size and position. The pelvic examination was negative. The nervous system showed nothing unusual. The urine was acid, specific gravity 1.021, otherwise negative. The blood examination showed hemoglobin 68 per cent; red blood cell count, 4,800,000; white blood cell count, 5,900; polymorphonuclears, 46 per cent; large lymphocytes, 13 per cent; small lymphocytes, 41 per cent. The Wassermann and Kahn reactions were negative. The blood pressure was 115/88. The metabolic rate was minus 2. The Vandenbergh reaction was negative and the icterus index was 5. The blood sugar was .09 mgm. per 100 cc. X-ray findings of the chest were negative.

The patient worked as a stenographer up to three weeks before coming to my office. On account of the marked asthenia she was moved to the hospital. Here the asthenia rapidly grew worse, dyspnea developed and the paient died five days after admission to the hospital.

The pertinent findings at the postmortem examination, performed by Dr. Walter M. Simpson, Pathologist, Miami Valley Hospital, follow: The body was that of

a fairly well developed adult white woman. The skin showed diffuse deep brownish pigmentation with multiple scattered, more deeply pigmented areas, averaging pinhead size. The pigmentation was most marked in the axillae and perineum. The sclerae showed no pigmentation. There was slight, soft, symmetrical enlargement of the thyroid gland. The mucous membranes showed marked pallor with diffuse brownish pigmentation, less marked than in the skin.

Because of stated restrictions the brain was not examined.

The thymus showed marked hyperplasia, measured 8 x 6 x 1.3 cm., and weighed 38 grams; on section, there was no evidence of fatty atrophy (see fig. 1). The heart was much smaller than the cadaver's right fist. The aortic orifice barely admitted the tip of the thumb. The lungs were negative save for intense acute passive congestion and scattered areas of healed tuberculosis. Multiple healed tubercles were found in the bronchial nodes. The thoracic aorta showed marked hypoplasia. The thyroid gland showed an excess of colloid.

The spleen was approximately twice normal size; it weighed 319 grams; on section, there was a marked increase in the size and number of the Malpighian corpuscles. There was a marked increase in the number and size of the solitary lymph follicles of the intestinal tract; the Peyer's patches of the small intestine showed marked lymphoid hyperplasia. The mesenteric and retroperitoneal lymphnodes showed distinct hyperplasia, varying in size from kidney bean to cherry.

The left adrenal was found after considerable search in the renal fatty capsule; it was greatly reduced in size and on section showed no grossly visible medullary tissue;

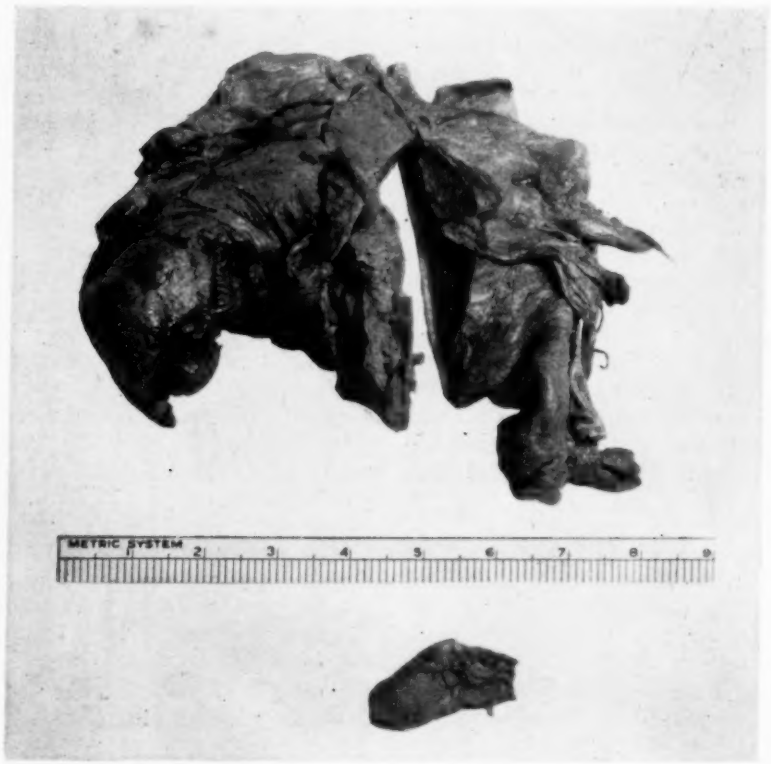

FIG. 1. Case 1. Persistent hyperplastic thymus and markedly hypoplastic left adrenal.

it measured 2.2x1x0.4 cm. (see fig. 1). Careful dissection failed to reveal the presence of a right adrenal; the tissue in the right adrenal region was saved for microscopic examination, in the hope of finding some evidence of adrenal tissue. The kidneys were of normal size, the fibrous capsule stripped readily and on section revealed only the deeply congested surface. The uterus showed marked hypoplasia; the other pelvic structures showed no abnormalities.

Microscopic examination of the hypoplastic right adrenal showed normal relationship of the cells of the three cortical zones; only a few medullary cells could be seen; there was no evidence of any inflammatory process. Careful microscopic examination of the tissue from the right adrenal region showed no evidence of adrenal tissue. The thymus showed abundant thymic tissue with many well-preserved corpsucles of Hassall. The thyroid tissue showed an excess of colloid; the acini were lined by a single layer of flat epithelial cells; there was extensive lymphoid hyperplasia of the thyroid tissue with many well-defined germ centers. Sections of the skin and vaginal mucous membrane showed an excess of melanin pigment in the basal layers of the epidermis.

Pathological diagnosis: Agenesia of right adrenal; extreme hypoplasia of the left adrenal. Persistent hyperplastic thymus. Generalized lymphoid hyperplasia. Marked hypoplasia of the aorta and heart. Status thymicolymphaticus. Diffuse melanosis of skin and mucous membranes. Terminal right sided cardiac dilatation with relative tricuspid and pulmonary insufficiency. Intense acute passive congestion of all organs. Healed tuberculosis of lungs and bronchial lymphnodes. Hypoplasia of uterus.

Case 2. A fifty-eight year old white woman was admitted to the Medical Service of the Miami Valley Hospital in a state of orthopnea. She gave as her chief complaints shortness of breath and anemia. She stated that she had been treated for some type of anemia for many weeks and had been sent to the hospital for transfusion. She said she was fairly well until four months before presenting herself at the hospital, at which time she first experienced difficulty in breathing, associated with marked weakness. Some difference in the color of her skin was noted, but no significance was attributed to it. The dyspnea increased and the patient was confined to her bed for only two days before coming to the hospital. She had experienced frequent urination, constipation, and sharp pains in the lower extremities. She was well developed but poorly nourished. A general melanosis was noted, especially emphasized on the exposed areas and approximating surfaces. The mucous membranes of the mouth and vagina showed similar melanosis. The respirations were deep and slow—eight to twelve per minute. The pupils reacted normally to light and in accommodation. The sclerae were clear. The few remaining teeth showed marked caries. The throat showed no marked inflammation. The tongue was rough and dry. There was no palpable cervical adenopathy. No evidence of active tuberculosis was found in either lung. The heart was small; the cardiac rate and rhythm were normal; a systolic bruit was heard at the apex. The blood pressure was 100-65. The liver and spleen were not palpable. An irregular nodular mass was outlined in both flanks in the position in which one would expect to find a ptotic kidney. There was no edema of the extremities. The pelvic and rectal examinations were negative, except for the presence of a small polypus which extruded from the cervical canal. The tendon reflexes were prompt and equal.

On entrance to the hospital, the patient had a temperature of 98.8° F., pulse 130. She lived but three days after entering the hospital, and during this time voided but twelve ounces of urine. This was acid in reaction; the specific gravity was 1.015; there was trace of albumin, a few pus cells, and an occasional hyaline cast. The blood examination showed hemoglobin of 80 per cent; red blood cells, 3,340,000; white blood cells, 28,150; and a relatively low lymph-

ocytosis. The Wassermann and Kahn reactions were negative. The blood chemical analysis showed creatinine content of 15 mgm. per 100 cc., urea nitrogen 60, urea 128, and blood sugar 143. These greatly increased nitrogenous values were coincident with marked asthenia, increased melanin pigmentation, dyspnea, constipation, and hypotension. A diagnosis of adrenal insufficiency was made, probably due to the pressure exerted upon the adrenals by the two masses found in either flank, or to neoplastic invasion of the adrenals. A further diagnosis of renal insufficiency was made on the basis of the blood chemical analysis.

The following abstract of the autopsy findings is taken from the report of the autopsy performed by Dr. Walter M. Simpson: The body was that of a tall slender white adult female. The skin was fine, soft and elastic and showed diffuse melanosis, most marked over the exposed areas, axillae and perineum. The mucous membranes showed approximately the same degree of melanosis as the skin. There was early cataract of the left lens; the sclerae were clear. The brain and spinal cord presented no noteworthy changes. The examination of the thoracic viscera revealed no abnormalities, except a moderate degree of aortic atherosclerosis and healed pulmonary and bronchial node tuberculosis; there was no grossly visible thymic tissue; the aorta presented no evidence of syphilis.

The liver was about two-thirds normal size, weighed 1100 grams and measured 22 x 16 x 15 cm. Through the capsule could be seen multiple thin walled cysts, many of which were elevated above the surface of the liver; these varied in size from pinhead to cherry and were filled with clear fluid. On section numerous similar cysts were found throughout the substance of the liver (see fig. 2). Otherwise the liver presented no abnormalities, except moderate cloudy swelling and chronic passive congestion. The left kidney was enormously enlarged, measuring 19 x 10 x 7.5 cm. (see fig. 3). The renal surface was very irregular due to the presence of thin walled cysts varying in size from small pea to walnut. On section, the entire kidney was seen to be made up of similar cysts, with small scattered islands of interposed renal parenchyma. The pelvis was thin walled and showed slight dilatation. The right kidney was slightly smaller but presented the same gross characteristics. The left adrenal showed marked pressure atrophy, being about one-third the normal size; it weighed 1.8 grams and measured 3 x 1.5 x 0.3 cm. The right adrenal showed practically the same degree of pressure atrophy; it weighed 2 grams and measured 3.2 x 1.4 x 0.4 cm. On section, the adrenals showed the normal relationship between cortex and medulla.

Microscopic studies of sections of the skin and vaginal mucous membrane showed a marked increase of melanin pigment. Sections of the liver showed multiple monolocular cysts lined by a single layer of flat epithelial cells; the liver cells showed patchy fatty degenerative infiltration with marked diffuse cloudy swelling and passive congestion. The adrenals showed marked atrophy of both cortex and medulla; there were no inflammatory infiltrations. The kidneys showed multiple large cysts lined by cuboidal epithelial cells; the glomeruli and tubules were widely separated by connective tissue, much of which was hyalinized and showed patchy lymphocytic infiltrations; there were numerous scarred glomeruli and multiple hyaline casts in the collecting tubules.

Pathological Diagnosis: Bilateral congenital polycystic kidneys. Renal insufficiency. Pressure atrophy of adrenals. Congenital polycystic liver. Diffuse melanosis of skin and mucous membranes. Moderately advanced aortic atherosclerosis. Healed pulmonary and bronchial node tuberculosis. Simple atrophy of pancreas. Polypoid glandular hyperplasia of endometrium. Multiple uterine fibromyomata. Cachexia.

Discussion

An interesting observation in the first case is that the patient was treated for one year for hyperthyroidism, although the blood pressure was 115/88

FIG. 2. Case II. Cut surface of polycystic liver, with many monolocular thin walled cysts, varying in size from pin-head to cherry, and filled with clear fluid.

and the metabolic rate was minus 2. The irritability of the heart, nervousness, and loss of weight were probably misleading. A prominent symptom in this case, common to many cases of Addison's disease, but rarely thought of as an important part of the clinical syndrome, was the marked back and chest pain. Early in the course of the disease, a diagnosis of catarrhal jaundice was made, but this was dismissed from consideration on the basis of the Vandenbergh test, icterus index, and absence of bile in the urine. A clinical diagnosis of Addison's disease was made because of the presence of marked asthenia, typical pigmentation and gastro-intestinal disturbances. The most striking pathological considerations in this case are concerned with the constitutional abnormalities. Hypoplasia of the adrenals is a common finding in status thymicolymphaticus; in this case, however, the agenesia and hypoplasia far exceeded that ordinarily observed in this diathesis.

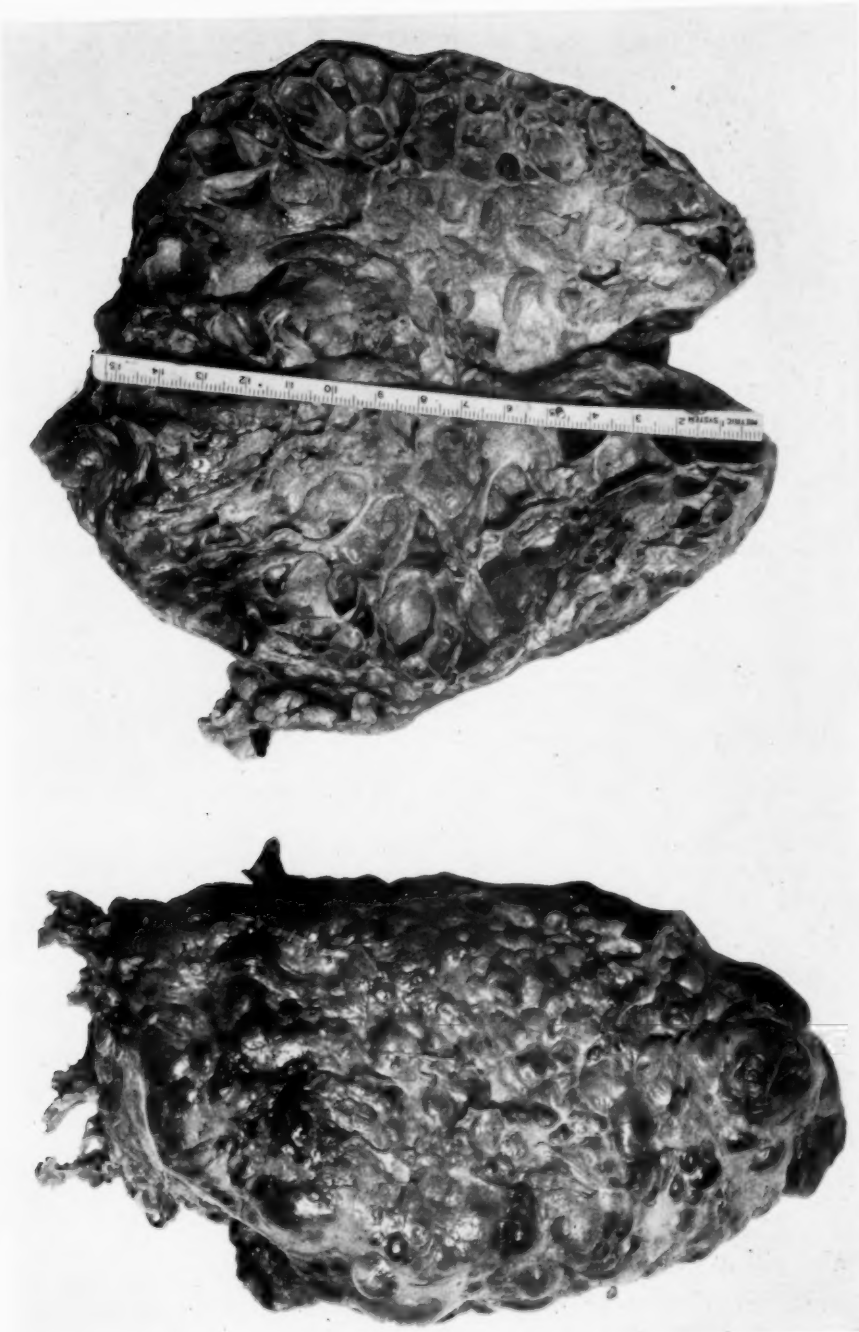

FIG. 3. Case II. Bilateral congenital polycystic kidneys, with multiple pea to walnut size thin walled cysts. The cut surface of the right bisected kidney and the capsular surface of the left kidney are shown.

The second case presented the four cardinal manifestations of Addison's disease—asthenia, hypotension, pigmentation, and gastrointestinal disturbance, associated with marked dyspnea. Although the patient was sent to the hospital for transfusion, the anemia was not an important consideration. Because of the age of the patient, 59, the possibility that the masses in the flanks were congenital polycystic kidneys was considered only to be dismissed. Despite the fact that terminal renal insufficiency usually occurs during early adult life in individuals with polycystic kidneys, the marked degree of nitrogen retention in the blood, associated with the bilateral nodular masses, should have more strongly suggested polycystic kidneys. It is noteworthy that pigmentation was not given consideration prior to hospitalization. The existence of hypotension in the presence of such marked nitrogen retention in the blood is worthy of special comment.

Summary and Conclusions

1. Addison's syndrome includes four cardinal clinical signs: melanosis, asthenia, hypotension and gastro-intestinal disturbances. Occasionally, hypotension is not encountered until late in the course of the disease.

2. Tuberculosis of the adrenals is by far the most common autopsy finding in cases of Addison's disease. In the remaining cases, syphilitic adrenalitis or neoplastic invasion are most often responsible for the development of adrenal insufficiency.

3. In two cases reported herewith, Addison's syndrome was related to agenesia and hypoplasia of the adrenals in one case, and to pressure atrophy of the adrenals produced by congenital polycystic kidneys in the other case.

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size thin walled cysts. The cut surface of the right bisected kidney and the capsular surface of the left kidneys are shown.

The History of Certain Medical Instruments

By Logan Clendening, M.D.,
Professor of Clinical Medicine, University of Kansas, Kansas City

GALILEO Galilei was attending a service at the Cathedral at Pisa (the date was circa 1581). He was not much interested in the sermon or the prayers, but his attention was directed to a chandelier which swung backwards and forwards above his head.

The lack of interest Galileo displayed in the blessed mutter of the mass that day in the Cathedral of Pisa has worked entirely to our benefit. Galileo was the scion of a noble Tuscan family and had become a student in medicine at the University of Pisa, much to his father's disgust, because the paternal desire was to make a cloth merchant out of him. He was to live to plague both his father and the dignitaries of all Italy, first by dropping weights from the leaning bell tower at Pisa, (which made him look like a fool to his father), and then by inventing a telescope with which "his Serenity and all the members of the Senate" (of Venice) could, after having "ascended at various times the highest bell towers in Venice to spy out ships at sea making sail for the mouth of the harbour," see them clearly, though "without my telescope they would have been invisible for more than two hours." His final outrage against public decency, of course, was to assert that the earth moved round the sun.

But all this was in the future. He was a medical student there in the Cathedral of Pisa when his roving eye lit upon that oscillating chandelier. It moved backwards and forwards like a pendulum in swings of ever decreasing amplitude. But what occurred to Galileo was that he thought he could calculate that even when the amplitude of the oscillation was the narrowest the time consumed in the small swing was the same as the time consumed in the long swing. This seemed queer: there was nothing like it in Aristotle he was sure: but was it true? Galileo had no watch. No one had a watch in those days. As he cast about for something to confirm his suspicions his fingers lit upon his pulse. Perhaps in the excitement of being on the verge of a discovery the young medical student felt his own heart beating and that suggested a timing piece.

At any rate, there he is—gazing upwards open mouthed, hand on wrist, while all about him the pious are crossing themselves and telling their beads.

Now! The chandelier swings—one—two—three—in three beats of his

*Read before the American College of Physicians, February 11, 1930, Minneapolis, Minn.

pulse. And back one—two—three. Now several minutes later. See the arc of the swing is much smaller now—you can see the corner of that window beyond its lefthanded excursion which you could not do before. But as to timing—one—two—three, and back—one—two—three. Just as before.

Galileo Galilei walked out of the cathedral into the bright sunshine which was then as now beating upon those white marble stones, and he began to think. He was by occupation a medical student so part of his thoughts concerned his pulse, this perfectly regular chronometer inside his body. There was food for thought there. But Galileo at the bottom of his soul was a mathematician. And mathematics had to do with time and weight and length of arc—all the things which seemed so mysterious about that chandelier. Heaven only knows how many people had seen chandeliers swinging in Cathedrals without ever having them suggest some relationship between those curious ponderables—time and weight. We in this age live very much by time. But the existence of time was less imminent in the days of Galileo. It was only because his mind was that of a natural mathematical genius that he began to analyze these relationships. Thus from Galileo's thoughts two ideas sprang—one concerning the timing of the pulse and one the relation of a pendulum's weight and arc to the period of its swing.

He went home and began to experiment. He tied a weight on a string and found that by exact measurements he had been right in the Cathedral. The pendulum swung through narrow arcs at the same rate of time that it did through wide ones. But he found that if he lengthened the pendulum it swung through its arcs at a slower rate. So much for the mathematicians.

Then the medical student came to the fore. He began comparing his own pulse under different conditions—after running and at rest—and found it varied. Then the pulses of his friends—old people and young people. There were variations here, too. So he constructed the first instrument with which to measure the pulse—Galileo's Pulsilogium. It was a very simple contrivance based on his string and weight idea. The string wound up on a wheel behind a dial. The dial had a pointer on it. When the pendulum swung synchronously with the patient's pulse the pointer indicated the rate at which that pulse was going.

FIG. 1. Galileo's Pulsilogium. The pendulum was synchronized with the pulse and then the dial pointed to the rate of the pulse. (From Harts' "Makers of Science," Oxford University Press.)

It was long before this idea of Galileo's began to be used as a practical thing in medical diagnosis. The age was not ripe. It is true Galileo's great contemporary, Kepler, used his pulse to record astronomic observations. And when Galileo went to the University of Padua as professor of mathematics in 1592 he doubtless interested the professor of medicine there, Santorio Santorio, usually known as Sanctorius. In 1625 Sanctorius published his comment on the first book of Avicenna and there describes a pulsilogium much like Galileo's.

Thus for the first time began the examination, and the counting of the pulse as an indication of the body's condition. It is today so fundamental and useful a procedure that it seems incredible it has been used for so short a time. Indeed, as we shall see, it is only within a hundred years that its significance became exact and valuable.

No wide use of the observation of the pulse as a method of diagnosis was made until the beginning of the eighteenth century. In 1707 a quaint little book called "The Physician's Pulse Watch" was published in England by a Staffordshire physician, Sir John Floyer.

Floyer says in the introduction to his book—"I have tried pulses by the minute in common watches and pendulum clocks and then used the sea minute glass."

We may imagine him starting to count a pulse and turning an hour glass upside down, counting the pulse until the sand had all run out. Science stumbles painfully along to its technical perfections.

"At last he was more happy. One Daniel Quare, a Quaker, had in the last years of the 17th century put on watches what Floyer called a middle finger, as we say a hand.

"Floyer's pulse watch ran 60 seconds and, you may like to know, can be had of Mr. Samuel Watson in Long Acre."

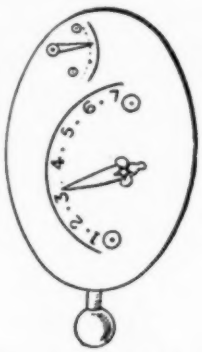


FIG. 2. Floyer's Physician's pulse watch. (From Dr. S. Wier Mitchell's Address—see Bibliography.)

"And now follow pulses of age and youth, pregnancy, exercise and sleep. And we learn how diet, blisters and the weather affect the pulse."*

For a time after Floyer's physician's watch was put on the market a vogue of wild speculation and theorizing about the pulse occured. "If any man," says Dr. Weir Mitchell, "wishes to nourish a taste for cynical criticism let him study honestly the books of the 18th century on the pulse. It is observation gone minutely mad: a whole Lilliput of symptoms: an exasperating waste of human intelligence. I know few more dreary deserts in medical literature from the essay on the "Chi-

(*Quoted from Mitchell—History of Instrumental Precision in Medicine).

nese Art of Feeling the Pulse," with which Floyer loaded his otherwise valuable essay, to Marquet's method of learning to know the pulse by musical notes, an art in which he was not alone. And error died hard. The doctrine of the specific pulse, a pulse for every malady, although rejected by de Haen, is in countless volumes, and survived up to 1827."

The next figure in the history of pulse counting is Robert Graves. You might see him often on the streets of Dublin a hundred years ago, a tall and distinguished figure, making his way to the Meath Hospital. Dublin was the great center of all European medicine for a period of several years at this time. And Graves was its most distinguished medical ornament. He had an interesting life behind him. While taking a walking tour in Austria during his student days he was arrested as a German spy. He proclaimed his British citizenship, as one of his ardently patriotic Irish biographers insists, with unconscious humour, but nevertheless his captors threw him into gaol because they insisted no one could speak as good German as he spoke and still be an Englishman.

Among his many contributions to clinical medicine was the practice he introduced of counting the pulse by the watch. And he put it on a more scientific basis than Floyer had. He made regular records and watched the outcome of his patients so studied. It was Graves who established the science of pulse counting so that, as Weir Mitchell says, "the familiar figure of the doctor, watch in hand, came to be commonplace."

The Thermometer

The exact measurement of heat also began, apparently, with Galileo, who also invented an air thermometer. It was a very delicate instrument, as it consisted of a glass tube of small bore dipping into an open glass of colored liquid, it was subject to barometric fluctuations, and was totally impractical to carry around so as to measure body heat. Later closed thermometers were developed, also by Galileo. Sanctorius, the Paduan, also used thermometers, and left illustrations of their use.

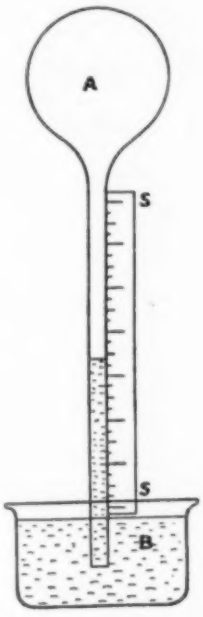
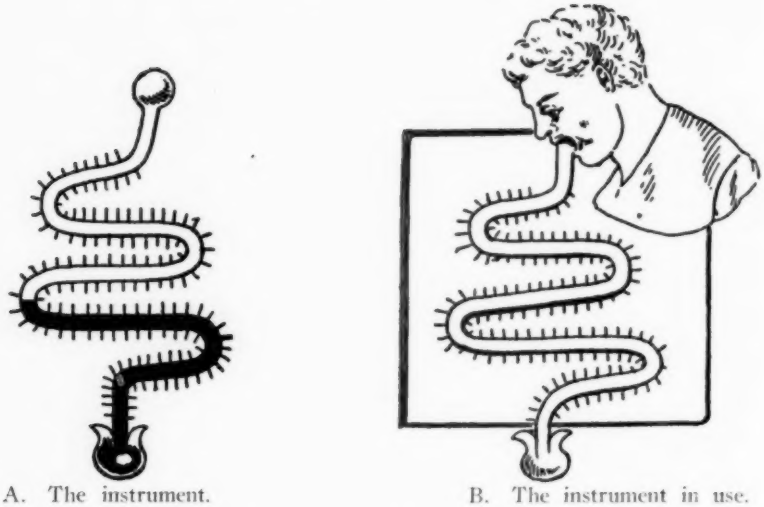


Fig. 3. Galileo's air thermometer. The mechanism—as heat expanded the air in the bulb A the liquid was forced downward in the tube. From Harts' "Makers of Science," Oxford University Press.)

When standards of measurement were proposed by Sir Isaac Newton and the familiar Fahrenheit, about



A. The instrument. B. The instrument in use.

FIG. 4. The first clinical thermometer. It was invented by Sanctorius, and is modelled on the air thermometer of Galileo.

1701 and 1714 respectively, they fixed as natural points the freezing point of water and the temperature of the normal healthy body. There were many errors due to the inaccuracies of early observation. They are too complicated to go into here and not germain to our discussion. Fahrenheit took the freezing point of water as zero, and the temperature of the body as 100. As we know the latter had to be changed so that now on the Fahrenheit scale it stands at 98 2/5.

The idea of the fluctuation of bodily temperature had been familiar since early times. But, of course, no exact record of the temperature of the body could be made until thermometers with a standard of measurement had been invented and generally introduced. The first work after Newton and Fahrenheit's establishment of a scale and the latter's invention of the mercury thermometer (which occurred about 1710) was done by a Scotchman, George Martine. In his "Essays and Observations" (1740) he made an extended comparative study of the matter. And his friend and fellow Scot, James Currie, who threw water on ship deck over his men with fever, adds to every one of his reports (published in 1798) on the use of water in fever a record of the temperature.

Napoleon had his temperature recorded. Dr. Archibald Arnott, Surgeon of the 20th Regiment, on April 21st, 1821, reported "as a result of the examination" of the Emperor's person he "could find no tension or hardness of the abdomen, the pulse was tranquil, the heat moderate." On April 3rd, "he passed a tolerably tranquil night, and slept a good deal, his pulse was 76, heat 96."

Certain ideas began to gain ground in the 19th Century. These were that in the normal body only the slightest variation of temperature occurred from time to time. Then there was

recognized a certain class of diseases—the fevers—which had an elevation of temperature. This knowledge was based on the recording of only one measurement of temperature—once in the course of the disease. Heart disease, dropsy, simple fractures, cancer, paralysis, dyspepsia were some of the diseases which did not have fever. Typhus, enteric, malaria, consumption, smallpox, hospital gangrene—some of the diseases which did have fever. Such was about the state of knowledge in 1850.

Around about 1850 a number of things began to occur. A young Manchester brewer read a paper at the British Association meeting at Oxford in 1847. The paper was on the subject of heat. The young man's name was Joule. His paper described how a falling weight made so much heat measured by having it fall on a baffle plate in a vessel of water. The queer young man named Joule seemed to think that the higher the height from which the weight fell the more heat there was produced. In fact he had it all figured out. The standard was 778 foot-pounds of work, the mechanical equivalent of the pound-degree Fahrenheit.

Joule had been expounding these ideas in Manchester for some time. He gave popular lectures on them. Like most popular lectures no one attended them. He finally managed to get on the program of the British Association, but since his ideas were evidently so wild, since he was not a *research professor*, and since he had given popular lectures, the chairman whispered to him to make the paper as brief as possible. No one seems to remember who that chairman was.

"Discussion," said Joule himself, describing the incident, "not being invited, the communication would have passed without comment if another young man had not risen in the section and by his intelligent observations created a lively interest in the new theory. His name was William Thomson."†

Fig. 5. Statue of Joule in Manchester.

Everybody up to that time had supposed heat was a substance—phlogiston. One of the great theorists of 18th Century medicine based his doc-

†Nearly half a century afterwards when Lord Kelvin unveiled a statue to Joule in his native Manchester (a statue I am happy to say, which shows him in a bathrobe and slippers) he said—"I can never forget the British Association at Oxord in 1847 when....I heard a paper read by a very unassuming young man who betrayed no consciousness in his manner that he had a great idea to unfold."

trine on this phlogiston theory. But these new ideas indicated it was a form of motion—of energy. How could a weight falling gain heat? Young Mr. Thomson, who discussed Joule's paper and who afterwards became Lord Kelvin, took the ideas of Joule and a forgotten Frenchman, Carnot, and established our modern ideas of the nature of heat and an absolute scale of heat and an absolute scale of temperature.

Thus, as she has done over and over in her history, medicine gained from another science, physics, an important conception of the nature of bodily heat. It was not to come to its fullest fruition for some time. Its first fruit was clinical thermometry.

The first application of this new knowledge of heat to sick people had to wait really a surprisingly long time for its development. When it came it was from the new German school of medicine and it is coincident with the rise of that method of teaching which still retains its supremacy to our own day.

The first great schools of medicine in Europe to which all students trooped were the Italian. In the Renaissance period when anatomy was the new and fascinating subject the Universities of Padua, of Bologna and of Pavia were great centers of liberal thought. In the later 17th and 18th Centuries the Italian universities divided honors with the schools of the Low Countries—Boerhaave was at Leyden, and Tulp at Amsterdam. British medicine was plainly in the ascendency from 1780 to 1830 with the London, Edinburgh and Dublin schools vying with each other. Then for a time the French under Laennec and Broussais and Louis and Trousseau succeeded them so that it was just as fashionable for a young man to go to Paris after completing his studies at home as, for instance, did Oliver Wendell Holmes and James Jackson, Jr., as it is now for him to go to Vienna.

But in 1840 there came to the chair of medicine at the University of Berlin a fat little boor named Schönlein. Schönlein's methods of teaching revolutionized medicine because he made the patient the center of all discussion. In other schools the teacher would enter the classroom, choose any subject he liked and treat the assembled students to an harangue on the subject more or less interesting or apt as the case might be. But not so Schönlein. He held his classes in the wards of the hospital. He had his chair placed by the side of a particular patient. Sinking into it he would hear the patient's story read: then he would discuss that story with the students and in German, not in Latin. Then the professor would rise and examine the patient; by looking at eyes, ears, chest, abdomen and listening with the stethescope: again sinking back in his chair he would ask the students to confirm his examination. Then the report on the analysis of the urine and other microscopical investigations would be read and discussed. A diagnosis of the cause and nature of the disease from which the patient suffered would be made, and methods of treatment explained. If the patient died, a postmortem examination of the body was made in the presence of the class and errors in diagnosis, if any, pointed out.

It was not long before this eminently practical method of teaching became famous. The universal introduction of the stethoscope and of percussion, of urinalysis, the perfection of the microscope, the rise of the science of chemistry—particularly the vanishing of scruples about the examination of the body after death—all made the intensive development of ti is method of clinical teaching particularly easy at this time.

But something else happened— a circumstance which has helped as much as anything to make clinical medicine in Germany continuously vivacious. There arose a group of brilliant men who adopted Schönlein's method and who became bitter and scornful rivals of each other. Happy is that medical school which has on its faculty three or more intensely vivid clinical teachers who hate one another and despise their rival's methods and views. Stimulating is the mental atmosphere of such an establishment.

After Schönlein's death he was succeeded at Berlin by two men, Frerichs and Traube, who were bitter enemies and intellectual opponents. They stalked by each other in the wards of the hospital followed each by his group of students without speaking, nor were even the students of one group allowed to speak to those of the rival.

So far as all this serves our account of the development of the thermometer into medicine, it is evident that in such an atmosphere of intense desire to make an accurate diagnosis every kind of a method was tried. Among them the thermometer. Traube was among the first to introduce it, beginning to use it in his clinic about 1850.

But the thermometers of those early days were not such as ours. They were nearly a foot long and required five minutes to make a record.

Doctor Lauder Brunton says:

"I had the appointment of house physician in the clinical wards of the Royal Infirmary at Edinburgh in 1866 to 1867.

"When I entered on my duties I found amongst other apparatus for use in the wards a case containing two clinical thermometers, one straight and the other somewhat bent. Each was

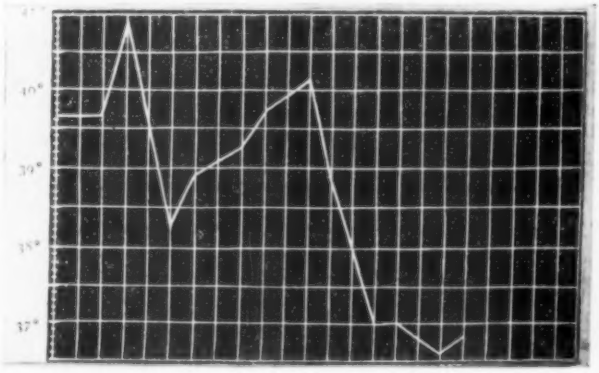


FIG. 6. The first graphic record of temperature, which was made by Ludwig Traube. (From Ebstein—see bibliography).

about ten inches or more in length and took about five minutes or more to reach the temperature of the body, when it was placed in the axilla. This thermometer case I used to carry under my arm as one might carry a gun."

Young Dr. J. S. Billings astonished his colleagues in the medical corps of the army of the Potomac by appearing on the field of battle with one of these gigantic temperature recorders. This was in 1862.

To illustrate the hardships of the early observers, Dr. George H. Savage recalls that about 1866 he was house physician for Sir William Gull in London. Sir William Gull instructed him and another house physician to take the temperature of a group of typhus fever patients every hour. As they had to sit by the patient for five minutes and bend over the mouth to read the thermometer (the thermometers were not self-recording such as the ones we use now and can shake down) they spent most of their time in close proximity to typhus patients, and Dr. Savage's associate came down with the disease himself.

These attempts were spasmodic and irregular. Only one record of a temperature in the course of any illness was generally taken—usually at the first visit.

But there was a hush of expectancy in the air of medicine about 1865 whenever the question of a patient's temperature was mentioned. Every wise clinician knew that there was something significant about it.

What was needed was some systematization of the procedure. It occurred to Dr. Carl August Wunderlich, who was professor of medicine at Leipzig, that possibly each febrile disease—each fever—had a characteristic *kind* of fever. He began to investigate. He laid down certain rules for himself. The thermometer must be convenient. It must record the temperature in a reasonably short time. It must be placed in some part of the body where it is entirely surrounded by body heat. The armpit, or axilla, was the favorite place selected by Wunderlich. But he also tried the mouth and the rectum.

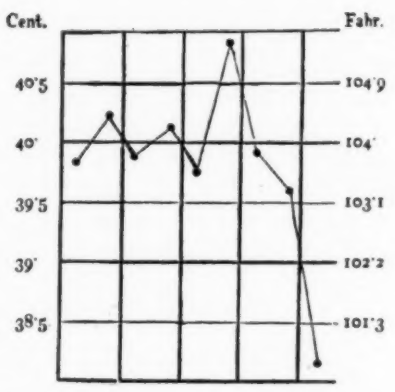


FIG. 7. Graphic record of temperature in Wunderlich's "Das Verhalten der Eigenwarme in Krankheiten."

Then in his large hospital he began to take temperatures every four hours on all patients. And before long he had plots and plans of temperatures which were just what he had suspected.

Here was a typhoid fever, for instance, carried along day after day—a slow rise in the beginning, a steady maintenance of fever for a week or ten days, then a slow daily drop—termination by lysis.

Here was pneumonia—entirely different—a rapid rise to dizzy heights—

a maintenance for seven or eight days —then a sudden drop—fall by crisis.

Here was tuberculosis—still another form—a monotonous day after day recurrence of low temperature in the morning and high temperature in the evening.

So, in 1868, Wunderlich published his results in a masterly book called in its English translation "Medical Thermometry" (Das Verhalten der Eigenwarme in Krankheiten). In its preface he gives all due credit to his predecessors—to George Martine and James Currie and Traube, as well as to all the workers in the pure science of physics, Galileo and William Thomson.

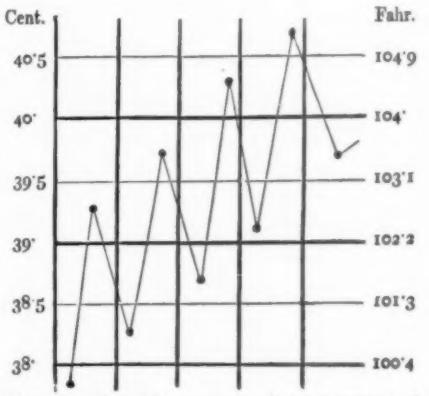


FIG. 8. Graphic record of temperature in Wunderlich's "Das Verhalten der Eigenwarme in Krankheiten."

Technical improvement in the thermometer occurred, too. The large ones were replaced by small neat affairs, registering the index on an ivory plate below which the bulb protruded one inch.

About the same time as the publication of Wunderlich's book Dr. Clifford Allbutt invented and introduced the small self-recording clinical thermometer much as we have it now.

THE SPHYGMOMANOMETER

The curate of Teddington in Middlesex from 1708 to 1761 must have been an interesting man. His name was Stephen Hales. He is one of the few clergymen one of whose sermons I should like to have heard. They were probably little filled with theological speculation, nor, I imagine, did the curate of Teddington, who was also rector of Porlock and of Faringdon, bother himself much with parochial duties. He would appear to have spent his life measuring the rise of sap in plants and the pressure of the blood stream in animals.

His ecclesiastical career is likely to puzzle an average American accustomed to seeing the vicars of God doing nothing but saving souls with great ardour unless one is familiar with "Barchester Towers." Here in the person of the Reverend Vesey Stanhope, who, though he had the livings of three cures, had resided for twelve years in Italy, you have the picture of the typical product of the Church of England system during at least most of the 18th and 19th Centuries. To this caste the Reverend Stephen Hales belonged. He was interested in Nature not in God.

He recorded his first observations on blood pressure in 1733, which, he says, were performed about "twenty-five years since," that is when he was first presented with his incumbency. They were performed on the crural arteries of dogs. Afterwards he experimented on "two horses and a fal-

low Doe." His first experiment on horses he records thus:

"In December I caused a mare to be tied down alive on her back; she was fourteen hands high, and about fourteen years of age, had a Fistula on her Withers, was neither very lean, nor yet lusty. Having laid open the left crural Artery about three inches from her belly, I inserted into it a brass Pipe, whose bore was one-sixth of an inch in diameter; and to that, by means of another brass Pipe which was fitly adapted to it, I fixed a glass Tube of nearly the same diameter, which was nine feet in Length. Then

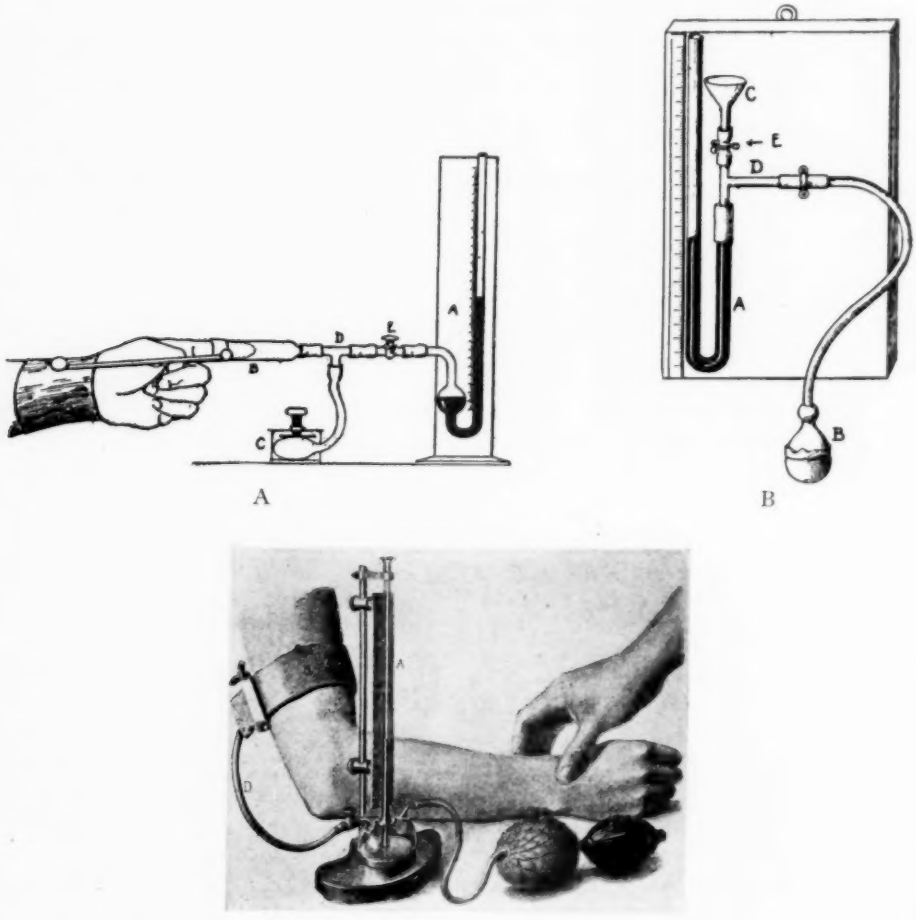


FIG. 9. The evolution of the sphygmomanometer.

A. Marcey's instrument—an early form.

B. Von Basch's syhygmomanomter as modified by Zadek. 1880.

C. The Riva-Rocci apparatus—1896.

(All figures taken from Janeway's "The Clinical study of the blood pressure." D. Appelton & Co.)

untying the Ligature on the artery, the blood rose in the Tube eight feet three inches perpendicular above the level of the left Ventricle of the Heart."

Following Hale's work the next advance in the study of blood pressure was the description of Jean—Leonard—Marie Poiseuille, in his graduating dissertation in medicine in 1828, of a hemodynamometer. This instrument measured the pressure by direct insertion of a cannula into the blood vessel, just as Hale's did, but it substituted a mercury column for the column of blood. Twenty years later, in 1847, Carl Ludwig added a float on the top of the mercury column and caused it to write on a recording cylinder, thus, as Stirling says, giving us at one coup "the kymograph or wave writer, and the application of the graphic method to physiology."

The first reading of the blood pressure in man was made in 1856 by Faivre, who again used the direct method, connecting an artery with a mercury manometer in the course of an operation. He recorded the blood pressure in the femoral artery as 120 mm. of mercury, and ni the brachial artery between 115 and 120 mm. Albert in patients undergoing amputations, made similar observations.

The idea of indirect measurement of blood pressure seems to have originated with Karl Vierordt, professor of physiology at Tübingen, in 1855. Marey applied this method to clinical medicine. The apparatus he used was based on the principle of the obliteration of the pulse, but was cumbersome inasmuch as the arm had to be placed in a glass box filled with water: this was connected both with an instrument to record the arterial pulsation and a mercury manometer to record the pressure within the glass box. Von Bosch, using the idea of indirect measurement, greatly improved the instrument by using a ball filled with water and connected with a manometer to obliterate the arterial pulse. This was still further improved by Potain, of whose portable sphygmamanometer Vaquez says—"for an entire medical generation it was as useful in research as the clinical thermometer."

Potain, von Bosch, Zadek and others made during this period the first regular measurements of the arterial blood pressure in man. They found it to be 130 mm. of mercury, but with variations between 110 and 160 mm. They noted measurements in patients with arteriosclerosis as high as 180 to 200 mm. of mercury and in fever patients 90 or 100 mm. These familiar figures, never improved upon or needing revision, show that the instrument was accurate and the observers painstaking.

In 1896 Riva-Rocci demonstrated an apparatus before the Italian Congress of Medicine which serves as the model for all of our present instruments. It consisted of a rubber bag or cuff which encircled the arm and was protected by an inelastic covering. This rubber bag was capable of having its internal pressure increased, and was connected with a mercury manometer. In 1897 Hill and Barnard substituted a calibrated pressure gauge for the mercury manometer. Every modern instrument for the reading of blood pressure clinically has followed the plans of these innovators.

THE HYPODERMIC SYRINGE

I cannot conclude this review without mentioning an instrument of therapeutic value. The history of the origin of hypodermic medication is confused. Garrison gives credit to Francis Rynd of Dublin for having "first employed hypodermic injections by a gravity device (of his own invention) for the relief of pain (1845-1861)." But

FIG. 10. Rynd's Hypodermic Syringe. (Photograph of the plate in the Dublin Quarterly Journal of Medical Science, 1861. XXXII, 13.)

Singer says—"No advance of this order compares in importance with the introduction of the Hypodermic Syringe by the ingenious French surgeon Charles Gabriel Pravaz (1791-1853)."

In 1836, however, another French physician, Lafargue, used a sort of needle trocar for introducing morphine under the skin in paste form. Previously an incision was made in the skin and the morphine placed in the wound. It is said that Doctors Taylor and Washington of New York in 1836 introduced morphine under the skin with a syringe of sterling silver with a leather piston: but an incision had to be made in the skin to allow the nozzle to be inserted. A cutting point on the needle was introduced by Dr. Charles Hunter of London in 1859.

What Pravaz did was to use a separate needle with a slip joint. In many French hospitals hypodermic needles are known as Pravaz. Apparently the first all glass syringes began to be made by Luer in 1896. Rynd's description of his syringe is as follows:

"The cannula (a) screws on the instrument at (b); and when the button (c), which is connected with the needle (f), and acted on by a spring, is pushed up (as in Fig. 2), the small catch (d) retains it in place. The point of the needle then projects a little beyond the cannula (Fig. 2). The fluid to be applied is now to be introduced into the cannula through the hole (e), either from a common writing-pen or the spoon-shaped extremity of a silver director; a small puncture through the skin is to be made with a lancet, or the point of the instrument itself is to be pressed through the skin, and on to the depth required; light pressure now made on the handle raises the catch (d), the needle is released, and springs backwards, leaving the cannula empty, and allowing the fluid to descend. If the instrument be slowly withdrawn, the parts it passes through, as well as the point to which it has been directed, receive the contained fluid and still more may be introduced, if deemed expedient.

"The subcutaneous introduction of fluids, for the relief of neuralgia, was first practiced in this country by me, in the Meath Hospital, in the month of May, 1844. The cases were published in the 'Dublin Medical Press' of March 12, 1845. Since then, I have treated very many cases, and used many kinds of fluids and solutions, with variable success. The fluid I have found most beneficial is a solution of morphia in creasote, ten grains of the former to one drachm of the latter; six drops of this solution contain one grain of morphia, and a grain or two, or more, may be introduced in cases of sciatica at one operation, with the very best effects, particularly if they are of long standing; or even in cases of tic in the head and face, with equally beneficial results. The small instrument is for operations on superficial nerves, the larger one for deep-seated nerves; for though it is not necessary to introduce the fluid to the nerve itself to ease pain, still the nearer to the seat of pain it is conveyed, the more surely relief is given. They were manufactured, and completed entirely to my satisfaction, by the celebrated surgical instrument-maker, Mr. Weiss, of London, and are faithfully represented in the accompanying lithograph, by Foster & Co., of this city."

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Editorials

THE BACILLUS-CALMETTE-GUERIN TRAGEDY IN LÜBECK

On the 27th day of July, 1929, Calmette sent a culture of the BCG-strain, Number 734, to Obermedizinalrat Dr. Altstaedt, Director of the Lübeck Gesundheitamt. With this same culture 573 children had been inoculated in France without apparent harm; and parallel cultures had been sent to Mexico and Riga and nothing unfavorable heard from them. In Lübeck the preparations for organization for a campaign in favor of inoculation with this culture, in the way of propaganda, lectures to physicians, instruction of midwives, etc., extended to February, 1930. In the meantime the culture was kept in the laboratory of Prof. Deycke in the Allgemeines Krankenhaus in Lübeck, and grown, at first, upon bile-potato medium, then later upon the Hohn egg-medium, in part upon hematin-egg medium, and at the last only upon the egg-medium. Transfers were made every four weeks by an experienced laboratory helper who had been with Deycke 17 years. The medium recommended by Calmette (synthetic fluid of Sauton) was not used. For the growth of the cultures and the preparation of the material for inoculation no completely separate compartment was used. No virulent human tubercle-bacilli were present in the incubators until September, 1929; the earlier cultures present having been killed by over-heating to 80° C. In September Deycke obtained a virulent strain of human tubercle bacilli, which was placed in the same compartment with the BCG inoculation preparations, and partigen was prepared according to the method of Deycke-Much, while the BCG cultures were kept exclusively in a smaller compartment of the inculator. As to the possibility of any mistake in the cultures, the BCG cultures were grown exclusively on *solid* media, the virulent strain on *fluid* media. On the day before the inoculation the material was tested for acid-fast organisms and found to be free from them. The inoculations were carried out in the children through the midwives exactly according to the French method, and the whole organization met the requirements of the French model. A dose (10 mg.) of the culture material as prepared for inoculation was given by mouth three times. About 50 per cent of all children born during the critical 60 days were inoculated, the total number of inoculated being 245. On the 17th of April, 1930, the first child died 33 days after the first inoculation. No autopsy was made. On the 20th of April, 54 days after the first inoculation the second child died. The autopsy showed a marked tuberculous process in the lungs, moderate organs. This case was regarded as an aerogenous infection, since the

mother of the child had tuberculosis but which was apparently not an open one. On April 25 and 26 autopsies made on the third and fourth children dying, who had been inoculated, left no further doubt that the deaths had been caused by a true feeding tuberculosis. Of 74 children inoculated before the 25th of March, 17 died, and a careful examination of 41 living showed evidence of tuberculosis in 28. The last death occurred on March 25. Inasmuch as there were 130 children inoculated later, up to April 26, further cases and deaths are to be expected. The clinical symptoms of the inoculated infected cases were: striking weakness and drowsiness after the inoculation, failure to gain in weight, meteorism, diarrhea, swelling of the cervical glands, and increase of temperature. In the more severe cases there was enlargement of the spleen, with roentgenologic changes demonstrable in the lungs. Some of the cases showed a characteristic cutaneous eruption, resembling pemphigus, which appeared after the second or third inoculation. The period of incubation in the 17 dead children was 4 weeks in 12 cases, 5 weeks in 3 cases and 3 weeks in 2 cases. On May 23, the incubation period in 47 still living children was estimated 3 weeks in 2 cases, 4 weeks in 10 cases, 5 weeks in 18 cases, 6 weeks in 11 cases and 7 weeks in 3 cases. In general, it was concluded that the prognosis was the more favorable, the longer the incubation period; but several children with a short incubation period ran a favorable course. The autopsy findings showed tubercles of varying size in all organs. A basal tuberculous meningitis was not present in any of the cases, although in one case a single tubercle was found in the leptomeninges. A most profound impression was made by these tragic events upon those in Lübeck directly connected with the immunizing attempt. In the height of his excitement Deycke on the 26th of April destroyed all that remained of the inoculation material in his laboratory. This on the face of it would seem to have been a most unwise act, as it prevented further study of the material used for inoculation. It may be accepted without doubt that the culture originally sent by Calmette was in truth avirulent; this would seem to be proved beyond all doubt by the harmlessness of its use in France and elsewhere. There are but two possible explanations of the Lübeck tragedy: Either there occurred in Lübeck *in vitro* a reversion of the culture to the virulent form, or the Calmette culture was either exchanged for or contaminated with virulent tubercle bacilli. It is not possible at this time to say positively which of the two alternatives is the true explanation. Against the first hypothesis may be urged the fact that up to the present moment it has been impossible to convert the avirulent Calmette culture into a virulent one. Numerous animal experiments have been carried out in this direction with guinea-pigs inoculated with the Calmette bacillus, in the attempt to lower the resistance of the inoculated animals by injury to the animal, mixed infection, addition of toxin, avitaminosis, chilling, etc. All of this experimental work failed to produce any increase of virulence in the Calmette organism. Further, in all of

the immunizing work that has been carried out upon animals with the Calmette cultures there has been no previous evidence of injury to any animal as the result of the inoculations. On the contrary, this experimental work has had favorable results in raising the resistance of the animals inoculated. For more than twenty years Calmette has busied himself with the problem of immunization against tuberculosis. On January 8, 1908, he for the first time began to cultivate a highly virulent form of bovine tubercle bacilli upon bile-potato medium, which after twelve years of cultivation upon bile-containing potato medium appeared to have completely lost its virulence. As the avirulence of this strain seemed to be fully established in 1921 he began most carefully to test it in children. In correspondence with his view that spontaneous tuberculosis infections arise through the gastro-intestinal tract he gave the prepared cultures in doses of 10 mg., three times, per os, to newborn children, before any possibility of infection from the environment could take place. In children exposed to virulent tubercle bacilli he found an apparent protection from infection. The number of infants inoculated with the BCG cultures must now reach 300,000-400,000. Of these over 242,000 were in France. With the experience of such a material, any harmful effect of the inoculation, any virulence of the culture, or an acquired virulence in the bodies of the inoculated children would certainly seem to be wholly excluded. Even if such occurred in only a relatively small per cent of the inoculated, the number of such would reach 4000 or more cases, and such a number of unfavorable results could not be kept concealed. In truth, no such harmful effects have ever been observed. It can readily be seen, therefore, what a catastrophe the tragic events in Lübeck must be to Calmette and his adherents. Naturally a storm of discussion has been aroused by the sad occurrence, especially in Germany where physicians were very slow to take up the Calmette work. Indeed, the direct stimulus to the Lübeck experiment was a personal letter from Calmette, accompanied by a culture of the BCG strain, written in July, 1929. In fact, a certain reproach against Germany's slowness to accept the Calmette work, had already been heard in other countries that had begun immunization work. It is but natural that the uncertainty as to the cause of the Lübeck fatalities should lead to a marked set-back to the Calmette work; but, as expressed by Professor Lange of the Reichgesundheitamt, the disastrous results at Lübeck offer no sufficient grounds for an unfavorable judgment of the BCG method of immunization.

Abstracts

The Hormone of the Adrenal Cortex. By FRANK A. HARTMAN and KATHERINE A. BROWNELL (Proc. of the Soc. f. Exper. Biol. and Med., June, 1930, p. 938).

These authors have previously demonstrated that an extract which will definitely prolong the lives and ameliorate the symptoms of adrenalectomized cats can be made from adrenal cortex. They have proposed the name of cortin for this hormone, which is essential to life. Heat (80° C. for 5 minutes) destroys it. It is lost upon repeated precipitation with NaCl. Therefore some other method of concentration must be employed. An extract of any desired concentration can be obtained by extracting the cortex with ethyl ether. After removing the ether *in vacuo* the residue is extracted with warm 80% alcohol. Chilling precipitates much inactive material. Removal of the alcohol *in vacuo* is followed by the extraction of the residue with water to make the desired concentration, or extraction by alcohol is repeated for further purification. Completely adrenalectomized cats treated with this extract not only live indefinitely in good condition, but are also able to meet unusual demands as well as normal animals. They can undergo major operations, and the wounds heal promptly. They seem to resist infections to which untreated adrenalectomized cats often succumb. One cat had an abortion following the removal of the second adrenal, and bled for several days afterward. Yet by the use of this extract she recovered. Another cat was etherized and thoroughly explored for accessory adrenals 136 days after the removal of the second adrenal, with recovery as prompt as would be expected in a normal cat. If adrenalectomized cats are given more extract than is necessary to keep them in fair condition they eat more and gain in weight. Blood urea remains within the normal range. One adrenalectomized cat has been rescued from the final stage of prostration due to an inadequate supply of cortin 3 times by injection of extract. The last time dyspnea and convulsive twitchings had developed. Seventy minutes after the injection of the extract the cat was sitting up. In 85 minutes she was shivering. In 100 minutes she was eating, not merely tasting, but taking her usual quantity of food. Individual animals show great differences in the amount of cortin which they require as well as the frequency of injection needed.

Some Metabolic Changes Occurring in Prolonged Diathermy Treatments. By E. S. NASSET and S. L. WARREN (Proc. of the Soc. of Exper. Biol. and Med., June, 1930, p. 943).

Studies on the respiratory exchange and the sugar, non-protein nitrogen, chlorides and carbon dioxide content of blood, were made on anesthetized (morphine + amytal) dogs. Tracheotomy was done and connection made to a Benedict universal apparatus. Blood analyses were done by standard methods. The high frequency current had the following characteristics: wave length — 200 meters, relatively high voltage, currents from 500 to 1000 milliamperes. Electrodes were placed on the left upper arm and right thigh, or on either side of the head. Treatment continued from 1 to 3 hours. Temporary measurements were made with thermocouples and mercury wavometers. The respiratory metabolism invariably increased, in some cases 150 per cent. Body temperatures were elevated 5 to 7° C. When blood sugar was initially relatively high there was a gradual depletion during diathermy; in cases of low initial concentrations a preliminary rise was noted followed by a fall. The end result was a marked hypoglycemia (30 to 50 mg. per 100 cc. blood). Non-protein nitrogen was in some cases increased to 200 per cent

of normal. Chlorides failed to exhibit any gross changes. The carbon dioxide content of whole blood and plasma invariably dropped to a rather low level (about 35 vol. per cent). Panting was induced in some animals, during which time the respiratory rate exceeded 250 per minute.

Experimental Polyneuritis in Chickens Given Jamaica Ginger. By J. H. WATKINS (Proc. of the Soc. f. Exper. Biol. and Med., June, 1930, p. 900).

Four apparently healthy chickens weighing approximately one kilo each were used in this experiment. Jamaica ginger obtained from a community where there were many patients with peripheral neuritis was used. Two of the chickens received daily doses of 2 cc. fluid extract of ginger and the other 2 received an equal amount of 83 per cent ethyl alcohol. The chickens were kept in a cage out of doors and given a diet of cracked corn and oats. Thirty-eight days after receiving the first dose of ginger and after each chicken had been given a total of 70 cc. there was no signs of muscular weakness. There had been some loss of weight during this time. On the 39th day the two chickens receiving ginger showed slight motor weakness and loss of coördinating power in both extremities. Feeding ginger was discontinued at this time. During the next two days there was a progressive motor weakness and the difficulty in walking or standing was marked. Control of the feet was wholly lost, and the toes were turned under the feet when an attempt was made to walk. The legs rather than the feet were used to support the body when resting. There was apparently no sensory disturbance of the extremities and no edema was observed. A thick suspension of rice polishings was given through a tube when the paralysis was first observed and daily thereafter for a period of 5 days without any improvement. One of the chickens died 8 days after the onset without any improvement. This investigation is being continued with a larger number of chickens, employing ginger with phenol, ginger without phenol and ethyl alcohol. At the 9th day after the experiment was begun only those receiving phenol ginger showed any leg weakness.

Effect of Sodium Salicylate on Intradermal Reactions of Rabbits. By O. E. HAGEBUSH and R. A. KINSELLA (Proc. of Soc. f. Exper. Biol. and Med., June, 1930, p. 922).

Sodium salicylate is commonly used in the treatment of infections, especially those presumed to be due to invasion by streptococcus. The old idea that acute rheumatic fever is due to infection by streptococcus, and the recently developed conception that the disease is involved in a process of allergy to streptococcus, stimulated this study of sodium salicylate in relation to allergy to streptococcus. Rabbits, inoculated with cultures of a strain of *S. hemolyticus* of low virulence, were used for this study. Following the intra-cuticular injection of streptococci, purulent arthritis invariably resulted and persisted until the death of the animal. In the first series of animals 15 controls gave strongly positive intradermal reactions 10 days after the production of arthritis, and 8 animals, given sodium salicylate 24 hours before the production of arthritis and at 24 hour intervals thereafter showed slight or no intradermal response. In another experiment, 16 control animals gave strongly positive reactions; 28 animals receiving sodium salicylate, gave slight or no reactions. 4 animals receiving glycin alone gave strongly positive reactions; and 16 animals receiving mixture of glycin and sodium salicylate gave strongly positive reactions. From this work it seems possible to draw the following conclusions: Sodium salicylate suppresses the allergic dermal reactions of rabbits to filtrates of hemolytic streptoccoccus. This effect is most definite when sodium salicylate is given before the focus of infection has developed. There is no relation between the presence or absence of this dermal reactivity and the character of the vascular pathology.

Primary Carcinoma of the Lung. By PAUL D. ROSAHN (Amer. Jour. of the Med. Sc., June, 1930, p. 809).

The postmortem incidence of primary carcinoma of the lung is steadily increasing, and this increase is real and absolute. Combined statistics show that primary carcinoma of the lung at autopsy from 1910

to 1919 comprised 0.44 per cent of autopsies, and 4.39 per cent of all cancers. Since 1920, primary carcinoma of the lung comprised 0.89 per cent of autopsies and 6.98 per cent of all cancers. Primary cancer of the lung is not as rare as was formerly believed. Because of its increased frequency, the clinician should give this affection serious consideration in differential diagnosis in patients of the carcinomatous age presenting puzzling lung symptoms and signs. An early diagnosis will permit accurate prognosis, and in selected cases, perhaps, surgical therapy.

Observations on the Possibility of Methyl Chloride Poisoning by Ingestion with Food and Water. By W. P. YANT (Public Health Reports, May 9, 1930).

The danger of life from the escape of noxious or inflammable refrigerating media into the air is being given considerable attention in the design and installation of mechanical refrigeration devices. In addition to atmospheric contamination and possible poisoning by inhalation, however, attention must also be given to possible contamination of food and poisoning by ingestion. In the present popular design of these devices the cooling mechanism is situated inside the comparatively air-tight cabinet with the food, and small leaks, which might be insignificant from the viewpoint of appreciable contamination of the external atmosphere, would create high internal concentrations. While there is no definite evidence that food poisoning has occurred or that this type of hazard exists with the refrigerants in current use, nevertheless, the possibility is a matter of concern to manufacturers of refrigerating devices and products, to health officials and to the public. The Bureau of Mines, with the cooperation of manufacturers of methyl chloride (CH_3Cl) has been engaged in the study of acute and chronic poisoning resulting from exposure to contaminated air. This work has been extended to include poisoning by ingestion. The possibility of poisoning by ingestion of methyl-chloride contaminated food and water was studied by exposing dogs. No apparent signs of poisoning were caused by the average daily ingestion on four successive days of 550 grams of ground raw beef or 200 cc. of milk that had been exposed 15 to 75 hours to 100 per cent methyl-chloride vapor at 35° F. No apparent symptoms of poisoning or changes in the hemoglobin and blood cells were caused by the ingestion of methyl-chloride contaminated water on 115 days of a total period of 171 test days. Also, no formates were found in the urine. Autopsy and examination of frozen sections, however, revealed a moderate degree of intracellular fatty degenerative infiltration affecting the ascending, descending and collecting tubules of the kidney. The glomeruli and convoluted tubules were apparently undamaged. Analysis showed the water to be 75 to 100 per cent saturated with an average methyl-chloride content of 0.595 gram per 100 cc. of water. This was the only water given the animals on six days of each week of the test. The taste of water saturated with methyl chloride at 68° F. is sharp, sweetish, and sickening when first taken into the mouth, followed almost immediately by a burning sensation. Persons would not drink more than a mouthful or two. It was frequently refused by the animals, even though they were deprived of other water.

Ursache und Bedeutung der post-operativen Acidose. By E. RAAB and F. WITTENBECK (Klin. Wochenschr., February 9, 1930, u. 255).

After narcosis and operations variations in the acid-base balance take place. Since Criele in 1917 called attention to these changes they have been generally spoken of as a postoperative acidosis, when a lowering of the alkali reserve and increase in the hydrogen-ion concentration of the blood occur. In surgery the postoperative acidosis has received especial attention from many sides, and it has been generally regarded as a dangerous complication in the course of healing. Above all the American school has held the postoperative acidosis to be dangerous. This conception rests in part upon the fact that Reimann and Bloom found in numerous investigations a marked increase in ketone bodies in the blood. Their value often rises after operation to 225 mg. This

increase in ketone bodies has been generally held responsible for the lowering of the normal blood reaction. Futher, a postoperative rise in the blood-sugar has been observed by other workers; and both phenomena have been regarded as related, namely, an increase in the blood-sugar goes hand in hand with an increase in the H-ion concentration. These postoperative phenomena affect the nitrogen metabolism, as has been demonstrated. For the neutralization of the acidosis the bicarbonate of the blood and the free ammonia given up from the liver was utilized. In the urine there occurs an increase in the ammonia value. Since these phenomena have never been completely studied in one and the same patient, but their study has been confined to scattered observations in one or the other metabolic directions, and in part studied on animals, and because of various contradictions, the authors undertook in their operations to study the cause and degree of the metabolic disturbances after operation and narcosis in human subjects. This was done in the case of nearly 100 patients. The hydrogen-ion concentration and alkali reserve were studied according to the methods of Hasselbach, Straub and Meier; the blood-sugar according to Hagedorn-Jensen; acetone and Beta-oxybutyric acid according to Engfeldt; ammonia according to Folin. As a result of their study the authors conclude that a number of factors are responsible for the postoperative disturbances of metabolism. A postoperative acidosis can sometimes arise through damage to the liver from inhalation narcosis. Disturbances of the hepatic function leads to pathologic products of metabolism, and to an increase of the acidity of the blood. The operation shock alone can, through the stimulation of the splanchnic, produce the same metabolic disturbances. Further, the psychical alteration of the patient before the operation is of significance in the postoperative metabolic changes. The effects of the psychical excitation may be manifest before the operation. Further, the overacidity of the organism through carbonic acid must be taken into consideration, due to the lowered excitability of the respiratory center through the anesthetic. When the paralysis of the respiratory center through the narcosis ceases, the acids of the blood again exert their full effect upon the respiratory center. This leads to an overventilation and to an increased output of carbonic acid through the lungs. So in many cases there will result an alkalosis of the blood although ketone bodies are present in the blood in increased amount. Finally, the hunger-state associated with the operation with its acidosis can increase the postoperative disturbances of metabolism. The administration of sodium bicarbonate can produce a normal blood reaction, but it has no influence on the cause of the metabolic disturbance. It should be emphasized that an alkalosis may be present after the operation. It follows that the methods of combatting the postoperative acidosis are useless, and may be even harmful. The authors insist that it is not necessary to treat the postoperative acidosis. In 100 patients they have never seen any damage to the organism due to the metabolic disturbances mentioned above. The body is always in a position to compensate for the postoperative disturbances of metabolism. They conclude with the statement that *the postoperative acidosis has no significance, and has no unfavorable influence upon the postoperative course. To avoid more marked metabolic disturbances after the operation the patient should have the least possible preparation for the operation. If any disturbance in the postoperative state occurs its cause is never to be sought for in postoperative acidosis.*

Reviews

Varicose Veins. *By* H. O. McPheeters, M.D., F.A.C.S., Director of the Varicose Vein and Ulcer Clinic, Minneapolis General Hospital; Attending Physician New Asbury, Fairview and Northwestern Hospitals, Minneapolis, Minn. 233 pages; illustrated with half-tone and line engravings. Second revised and enlarged edition. F. A. Davis Company, Philadelphia, Pa. Price in cloth, $3.50 net.

The frequency of varicose veins and ulcers and the unsatisfactory nature of the treatment usually accorded them are undoubtedly responsible for the interest excited by McPheeters book, the second printing of which was exhausted in less than five months. A new edition has now been prepared to meet the demands created by this interest. In it an attempt has been made to make more clear certain points that were not sufficiently understandable by the general practitioner. Particularly in the case of the Trendelenburg test, both as to its demonstration and clinical application have so many inquiries been made that it has seemed best to present it in a separate chapter. The general technic is practically unchanged, although slight modifications developed with greater experience have been made in it. The work on the pathological changes following the injections has been continued by the study of biopsies made at intervals of one hour to two years following the injection. The author states his views clearly and concisely.

Physiology and Biochemistry in Modern Medicine. *By* J. J. R. Macleod, M.B., LL.D., D.Sc., F.R.S., Regius Professor of Physiology in the University of Aberdeen, Scotland; Formerly Professor of Physiology in the University of Toronto, Canada, and in the Western Reserve University, Cleveland, Ohio. Assisted by R. G. Pearce, A. C. Redfield, N. B. Taylor, and J. M. D. Olmsted, and by others. Sixth Edition. 1074 pages, 295 illustrations, including 9 plates in color. The C. V. Mosby Company, St. Louis, Missouri, 1930. Price in cloth, $11.00.

During the three years since the appearance of the last edition there have been no important discoveries in the field of physiology and biochemistry. There has been, however, a steady increase in general knowledge. This has been recognized, in the preparation of the present edition, in various changes and additions that have been spread throughout the volume. Material that is out of date or no longer considered necessary has been omitted. New matter has been put in its place, or has been placed in small print. The size of the book therefore, remains unchanged. It remains one of the best textbooks on physiology that has yet been written, and gives a very complete survey of the science of physiology as it stands today. This book is heartily recommended to medical students.

Trauma, Disease, Compensation. A Handbook of Their Medicolegal Relations. *By* A. J. Fraser, M.D., Chief Medical Officer, Workmen's Compensation Board, Winnipeg. 524 pages. F. A. Davis Company, Philadelphia, 1930. Price in cloth, $6.50.

The increasing importance of workmen's compensation makes this volume of interest to medical men in Canada and America, inasmuch as the number of industrial casualties in these two countries reaches enormous figures, even greater than the casualties of the War. Out of the large number of injured workers grow many problems which almost daily meet the medical and surgical practitioner, and are finally decided by Compensation Boards, which in the reviewer's experience, are all too often ignorant and incompetent. Before such Boards opinions based upon scientific knowledge are often set aside by the judgment of an ignorant

and prejudiced Commissioner. If there is anything in the United States needing a thorough renovation from the bottom up it is our methods of taking testimony in Compensation cases; and in settling such claims. The workman and his relatives are usually favored in the grossest way, and the opinions of ignorant and inexperienced physicians are credited over those of scientific men of repute. The present volume has been prepared in the hope that a useful purpose will be served by assembling the opinions of representatives teachers and writers in the medical field on the difficult subject of the influence of trauma in giving rise to subsequent conditions of disease. Such information as exists upon this subject is scattered through the literature, and the statements made on this point are usually vague and casual. The opinions quoted are in the main verbatim. Every care has been taken not to distort the meaning of the writer quoted. The compilations of reputable and expert opinion on the question at issue will, it is felt, furnish a useful encylcopedia on a subject not heretofore covered in a general and comprehensive manner; and should aid medical men and industrial boards in arriving at an adjustment of debatable medical problems in this field. No claim is made for original work in the preparation of this volume. The work has been largely that of an editor in assembling and arranging the material selected, and the author assumes no responsibility for the opinions set forth. If medicolegal testimony in the field of industrial compensation is to be put on a decent basis in this country, reference book of authority will constitute the foundation for such, and this volume is a step towards the securing of that foundation. It is however, very superficially done, and the range of literature covered by it is very limited. It is a pity that the editor-author could not have had access to the extensive German literature on the subject.

The Normal Diet. A Simple Statement of the Fundamental Principles of Diet for the Mutual Use of Physicians and Patients. *By* W. D. Sansum, M.S., M.D., F.A.C.P., Director of the Potter Metabolic Clinic, Department of Metabolism, Santa Barbara Cottage Hospital, Santa Barbara, California. Third Revised Edition. 134 pages. The C. V. Mosby Company, St. Louis, Mo., 1930. Price in cloth, $1.50.

The author has for many years given the subject matter of this book in lecture form to patients suffering from various nutritional disorders. He believes that errors in diet are very common, and that such errors are responsible for many minor ailments as well as some of the more serious ones. He, therefore, believes that a simple statement of the fundamental principles underlying the selection of a normal diet may fill a definite need. There are eight chapters dealing respectively with the bulk requirement of the body, the acid-ash type of acidosis, the acetone type of acidosis, the caloric requirement of the body, the protein requirements of the body, the mineral requirement of the body, the vitamin requirement of the body, and the water requirement of the body. Chapter IX is given up to Diet Menus. The book is a common sense one, and free from the fads that characterize the average book on diets.

The Treatment of Skin Diseases. In Detail. *By* Noxon Toomey, M.D., B.A., F.A.C.P., Late Instructor in Dermatology, St. Louis University. Volume Three. 512 pages. The Lister Medical Press, St. Louis, 1930. Price, $7.50.

The methods of treatment described in this book have been adequately experienced by the author in his private and dispensary work of the past fifteen years; and are the methods employed by him at the present time, as they appear to be the most advantageous. In the majority of instances, however, alternative methods are described in order to meet the exigencies of practice in communities where some drugs and some physiotherapeutic facilities are not likely to be immediately available. The book includes an adequate description of the treatment of all known skin diseases. It constitutes Volume Three of the author's "Principles and Practice of Dermatology", the other two volumes being I, Pathology and II, Diagnosis. The author's object has

been to present the therapeutics of skin diseases in a form originating out of his own experience; and the book is no mere rewording of what has been written by others on the subjects treated. In the case of only a very few diseases, mostly tropical, has he been obliged to fall back upon the published observations of other physicians. For those he makes due acknowledgements. The completeness of the discussions and the colloquial manner of treatment are in the author's opinion reasons justifying the publication of this book. In addition it contains some original contributions to the treatment of skin diseases. The claim is made that there is at present no text of like thoroughness on cutaneous therapeutics.

Recent Advances in Diseases of Children. By WILFRED P. PEARSON, D.S.O., M.G., D.M., F.R.C.P., Physician in Charge of Children's Department, University College Hospital; Physician to Out-patients, Hospital for Sick Children, Great Ormond Street; Sometime Physician to Children's Department, Charing Cross Hospital; and W. G. WYLLIE, M.D., M.R.C.P., Physician to Out-patients, Hospital for Sick Children, Great-Ormond Street; Assistant Physician to Children's Department, Westminster Hospital; Assistant Physician to the Hospital for Epilepsy and Paralysis, Maida Vale. Second Edition, 548 pages, 20 plates and 34 text figures. P. Blakiston's Sons & Co., Inc., Philadelphia, Penna., 1930. Price in cloth, $3.50.

In this edition practically the same arrangement has been retained as in the first. A certain amount of revision and condensation has, however, been necessary because of the inclusion of new material. The main additions and alterations are concerned with Postvaccinal and Measles Encephalomyelitis; Chronic Infection of Tonsils and Adenoids; Causation of Cerebral Diplegia; Bronchiectasis, Thoracic Tuberculosis and Asthma; Chronic Abdominal Conditions; Congenital Syphilis; Skin Tests and Some Forms of Immunization. The present book, in spite of its title, is in reality a textbook of children's diseases. The aim of the authors has been to correlate problems of importance in children and adult, and to present investigations more particularly related to the child, in a manner useful to everyday practice, and to visualize children's diseases as a whole. They believe that a bare statement of the results of recent scientific research, as it affects the diseases of children would be both dull and disconnected. The intrusion of personal opinions is intended to be provocative, in order to get away from the habit of taking sets of symptoms—or a syndrome—as an isolated disease, disregarding the fact that many clinical variations may spring from a common basis. If they have systematized children into types unduly, it has been done intentionally, as the factors of "soil" and heredity cannot fail to influence the expression of morbid processes in different types. In order to get a true picture of disease in childhood the authors insist throughout their book that the clinician must have always in mind the grown-up child, the adult. This is a book on children's diseases written with a new and original slant, and, while including recent important additions to scientific knowledge in this field, it offers to the practitioner a consideration of the ailments of the young as they present themselves in every day practice.

College News Notes

Dr. L. J. Moorman (Fellow), Oklahoma City, was elected a member of the Executive Committee of the National Tuberculosis Association during its convention in Memphis, May 8.

At the annual meeting of the American Therapeutic Society at Detroit, June 20-21, Dr. Clement R. Jones (Fellow and Treasurer), Pittsburgh, was elected President for the ensuing year.

Dr. William C. Voorsanger (Fellow), San Francisco, was elected Representative Director for California of the National Tuberculosis Association, during its last annual meeting.

Dr. C. Ray Lounsberry (Fellow), San Diego, addressed the Dermatological Section of the California State Medical Association, which convened at Del Monte the last of May, upon the subject of "Dermatological Neuroses."

Dr. John G. Young (Fellow), Dallas, read a paper before the Dallas Medical Association at Mineral Wells during April on "The Effect of Infection Upon Peristalsis and Appetite with an Outline of Appetite Management."

Dr. Young is Chairman of the Dallas County Medical Milk Commission.

Dr. E. W. Gehring (Fellow), Portland, addressed the New Brunswick Medical Society at its 50th annual session at St. Andrews-by-the-Sea, June 24-25, on "Syphilis and What is Society's Attitude Toward It."

Dr. Samuel Goldberg (Associate), Philadelphia, was recently appointed Visiting Chief of the Pediatric Department of the Jewish Hospital.

Dr. Goldberg, with Dr. H. Brooker Mills (Fellow) and Dr. Kerman Snyder, is the author of a paper entitled "Pyloric Obstruction," which appeared in the April number of Medical Review of Reviews.

Dr. Benjamin Hobson Frayser (Fellow), Fort Harrison, Mont., is the author of an interesting article entitled "Medical Fraternities in North America," which appeared in the June Issue of Clinical Medicine and Surgery.

Dr. Samuel A. Levine (Fellow), Boston, addressed the Philadelphia Heart Association, May 7, on "A Clinical Conception of the Development of Rheumatic Heart Disease."

Under the direction of Dr. R. R. Snowden (Fellow), the Pittsburgh Diagnostic Clinic held an all-day program of lectures and demonstrations by the staff on April 30. "The subjects discussed were principally those which pertain to problems in diagnoses. Clinical methods and laboratory tests received equal consideration from the staff in an effort to evaluate and present a large number of diagnostic procedures."

Dr. Stewart R. Roberts (Fellow), Atlanta, was the invited guest who discussed "The Heart, Gall-Bladder Problem."

Dr. Thomas Klein (Fellow), Philadelphia, has been appointed Professor of Clinical Medicine at Temple University School of Medicine, while Dr. Allen G. Beckley (Fellow), Philadelphia, has ben appointed Clinical Professor of Medicine.

Dr. James Francis Rice (Fellow), Buffalo, is President for 1930-31 of the Buffalo Academy of Medicine. He served as Secretary of the Academy for four year, 1921-25.

Dr. A. B. Moore (Fellow), formerly of the Mayo Clinic, is now associated with Doctors Groover, Christie and Merritt in the practice of Roentgenology in Washington, D. C. Dr. Moore has also been appointed Professor of Roentgenology at Georgetown University. His Washington address is 1835 Eye St., N. W.

At the meeting of the American Medical Editors' and Authors' Association at Detroit on June 24, a resolution was adopted, urging legislature to provide for a Department of Health, headed by a medical secretary, in the Cabinet of the President of the United States.

Among members of the Board of Governors of the above Society the following members of the College were elected:

Dr. J. M. Anders (Master), Philadelphia
Dr. William Engelbach (Fellow), Santa Barbara
Dr. T. Homer Coffen (Fellow), Portland
Dr. Julius H. Hess (Fellow), Chicago
Dr. C. Ulysses Moore (Fellow), Portland
Dr. F. M. Pottenger (Fellow), Monrovia
Dr. W. Forest Dutton (Associate), Amarillo

Dr. John Hubeny Maximilian (Fellow), Chicago, is the Managing Editor of RADIOLOGY, the official publication of the Radiological Society of North America. Dr. Benjamin H. Orndoff (Fellow), Chicago, is the Associate Editor; Dr. W. Warner Watkins (Fellow), Phoenix, is an Assistant Editor; and Dr. William B. Bowman (Fellow), Los Angeles, Dr. L. J. Carter (Fellow), Brandon, Manitoba, and Dr. H. Kennon Dunham (Fellow), Cincinnati, are collaborators.

Dr. Kenneth M. Lynch (Fellow), Charleston, is the author of an article entitled "Education Versus Promotion," in the June Issue of the Journal of the Medical Association of Georgia.

In the June Issue of the American Journal of the Medical Sciences, the following Fellows contributed articles:

Dr. Cyrus C. Sturgis, Ann Arbor, with Dr. M. C. Riddle, "The Effect of Single Massive Doses of Liver Extract on Patients with Pernicious Anemia";

Dr. Soma Weiss, Boston, with Dr. J. E. F. Riseman, "The Symptomatology of Arterial Hypertension";

Dr. Edward L. Bortz, Philadelphia, "Viceroptosis: Its Clinical Significance and Treatment";

Dr. Paul A. O'Leary, Rochester, with Dr. Ruben Nomland, "A Clinical Study of One Hundred and Three Cases of Scleroderma."

Dr. Paul F. Whitaker (Fellow), Kinston, N. C., is the author of a paper on "Bronchiectasis" in the June number of the Virginia Medical Monthly.

Under the Presidency of Dr. C. Lydon Harrell (Fellow), Norfolk, the Norfolk County Medical Society held its annual meeting, June 2.

Dr. Harrell and Dr. Walter B. Martin (Fellow), Norfolk, were elected delegates to the next meeting of the Virginia Medical Society.

Dr. J. D. Willis (Fellow), Roanoke, is President of the Roanoke Academy of Medicine.

Dr. Thomas B. Futcher (Associate), Baltimore, was made President-Elect of the Association of American Physicians at their last meeting in May. Dr. James H. Means (Fellow), Boston, was re-elected Secretary.

Dr. Alfred L. Gray (Fellow), Richmond, was recently appointed Councilor of the Southern Medical Association from Virginia, the appointment being made by the President, Dr. Hugh S. Cumming (Fellow), Washington.

Dr. Coursen B. Conklin (Fellow), Washington, has been re-elected Secretary-Treasurer of the Medical Society of the District of Columbia for the year beginning July 1, 1930.

Dr. Kenneth M. Lynch (Fellow), Charleston, was installed as President of the South

Carolina State Medical Association at its last annual meeting. Dr. Edgar A. Hines (Fellow), Seneca, was re-elected Secretary.

The 31st annual meeting of the Association will be held at Greenville, S. C.

Dr. Henry Boswell (Fellow), Sanatorium, Miss., was elected President of the National Tuberculosis Association at its annual meeting at Memphis in May.

Dr. Stuart Pritchard (Fellow), Battle Creek, was elected one of the Vice Presidents.

Under the Presidency of Dr. Walter E. Vest (Fellow), Huntington, the West Virginia State Medical Association held its annual meeting at White Sulphur Springs, during the latter part of May. Dr. A. H. Hoge (Fellow), Bluefield, was elected one of the Vice Presidents for the succeeding year.

Dr. Vest was elected President of the Alumni Association of the Medical College of Virginia, during the annual meeting in May.

Under the Presidency of Dr. Henry Green (Associate), Dothan, Ala., the Chattahoochee Valley Medical and Surgical Association held its meeting at Albany, Ga., July 8-9.

Dr. George R. Callender (Fellow), Washington, was elected President of the American Association of Pathologists and Bacteriologists at the last meeting of the Association held in New York.

Dr. L. B. McBrayer (Fellow), Southern Pines, has been elected Managing Director of the North Carolina Tuberculosis Association.

Dr. W. S. Leathers (Fellow), Nashville, Professor of Preventive Medicine and Dean of the Vanderbilt Medical School, has been elected a member of the Board of Scientific Directors of the International Health Division of the Rockefeller Foundation.

Dr. Beverley R. Tucker (Fellow), Richmond, on the occasion of the anniversary of his birth, the twenty-fifth anniversary of his graduation in medicine and the fifteenth anniversary of the founding of the Tucker Sanatorium, was honored with a dinner given by Dr. Howard R. Masters and Dr. Asa Shield on April 26.

Dr. Joseph M. King (Fellow), Los Angeles, addressed the San Diego County Medical Society, June 10, on "Hypertension."

Dr. A. J. Carlson (Fellow), Chicago, was one of the speakers at the annual dinner of the Faculty and Alumni of Rush Medical College of the University of Chicago at Congress Hotel on June 10. During the meeting, it was announced that Dr. Frank Billings had endowed four Fellowships at Rush Medical College in the sum of $100,000. One of these Fellowships will be named in honor of Dr. Ernest E. Irons (Fellow), Dean and Professor of Medicine of the Rush Medical School.

Dr. Thomas B. Magath (Fellow), Rochester, was one of the speakers at the meeting of the Twin Lakes District Medical Society of Iowa on June 12.

In honor and recognition of twenty-five years of uninterrupted service in the medical school, Dr. Charles H. Neilson (Fellow), St. Louis, was tendered a dinner by the administrative board of St. Louis University School of Medicine on June 4. Dr. Neilson is Professor of Internal Medicine and Associate Dean of the school.

Dr. Robert S. Berghoff (Fellow), Chicago, was elected President of the Chicago Tuberculosis Society on May 20.

Dr. James H. Hutton (Associate), was installed as President of the Chicago Medical Society at its annual meeting on June 17.

Dr. Nathan S. Davis, III (Fellow), Chicago, was re-elected Secretary.

Dr. J. A. Myers (Fellow), Minneapolis, with Dr. H. D. Chadwick, presented a study on childhood tuberculosis before the Michigan Trudeau Society, June 10, at Battle Creek.

Dr. Meldrum K. Wylder (Fellow), Albuquerque, was elected President-Elect of the New Mexico Medical Society on June 5.

Dr. Walter A. Bastedo (Fellow), New York, has been elected President of the United States Pharmacopeia Convention.

Dr. Henry Kennon Dunham (Fellow), Cincinnati, was recently elected President of the Ohio Public Health Association.

At the annual meeting of the Pacific Northwest Medical Association at Butte, Mont., June 1-3, papers were presented by Dr. A. J. Carlson (Fellow), Chicago, "Involuntary Nervous System and Circulation"; Dr. George B. Eusterman (Fellow), Rochester, "Significance of Gastric Anacidity in General Medicine and Gastro-Enterology"; and Dr. Allen K. Krause (Fellow), Tucson, "Physiological Relations in Tuberculosis."

Dr. James D. Bruce (Fellow), Ann Arbor, will supervise the work in graduate medicine at the University of Michigan under a new Executive Committee to govern the University of Michigan Medical School, as recently appointed by the board of regents.

GIFTS TO THE COLLEGE LIBRARY

The following gifts of publications by members to the College Library are duly accepted:

Reprint:

Dr. Douglas Brown (Fellow), Washington, D. C., "Arthritis"

Reprint:

Dr. Philip B. Matz (Fellow), Washington, D. C., "Future Incidence of Nervous and Mental Disease Among Ex-Service Men"

Reprints:

Dr. Edwin Schisler (Fellow), St. Louis, Mo., "Anesthesia in Cardiac Disease and its Complications"
"Aneurysms"

Reprint:

Dr. Leonard F. C. Wendt (Fellow), Detroit, Mich., "Observations on a Summer Camp for Diabetic Children"

Dr. Sinclair Luton (Fellow), St. Louis, gave "Demonstrations of Interesting Heart Cases" on June 16 at the City Hospital, in connection with the program of the Clinical Conference by St. Louis Clinics, from June 9 to 21.

The following Fellows of the College attended the regular scientific conference of St. Luke's Hospital Staff on Friday, June 6, at the Union League of Philadelphia:

Dr. Carl V. Vischer, Philadelphia
Dr. G. Morris Golden, Philadelphia
Dr. E. J. G. Beardsley, Philadelphia

Dr. Beardsley (Fellow and Governor) was the speaker of the evening.

June 6 was the birthday anniversary of the late Dr. Carl V. Vischer, founder, leader and first Chief Surgeon of St. Luke's Hospital, Philadelphia (1896-1906). In honor to Dr. Vischer's memory, and as a tribute to his achievements, the scientific program was dedicated.

Dr. Linn J. Boyd (Fellow), New York, is the author of an article, "Symptomatology," which appeared in the June number of the Journal of the American Institute of Homeopathy.

The second annual round table of the Physicians Hospital of Plattsburgh, New York, was held August 22-23. Dr. William E. Robertson (Fellow), Professor of Medicine at Temple University School of Medicine, Philadelphia, gave an address on "The Present Status of Digitalis Therapy, which was discussed by Dr. Joseph Wolffe (Associate), Cardiologist at Temple University School of Medicine. Dr. Edward C. Reifenstein (Fellow), Professor of Medicine in the College of Medicine, Syracuse University, gave an address on "Some Phases of Syphilitic Heart Disease." Dr. Clarence H. Beecher (Fellow), Professor of Medicine in the Medical College of the University of Vermont (Burlington), gave a paper on "The Diagnosis of Pericarditis." Dr. Carl Wiggers (Fellow), Professor of Physiology, Western Reserve University Medical School (Cleveland), gave the Beaumont Lecture on "The Physiological Meaning of Common Clinical Signs and Symptoms in Cardio-vascular Disease."

EIGHTY-FIRST ANNUAL SESSION
of the
AMERICAN MEDICAL ASSOCIATION

Dr. William Gerry Morgan (Fellow), Washington, D. C., was inducted as President of the American Medical Association during the 81st Annual Session at Detroit, June 23-27.

Dr. E. Starr Judd of Rochester, Minn., a Fellow of the American College of Surgeons, was made President-Elect.

Among Fellows of the American College of Physicians who occupied executive or committee appointments during the Detroit meeting were:

BOARD OF TRUSTEES:
Dr. Rock Sleyster, Wauwatosa, Wis.
Dr. Allen H. Bunce, Atlanta, Ga.

JUDICIAL COUNCIL:
Dr. J. N. Hall, Denver, Colo.
Dr. James B. Herrick, Chicago, Ill.

COUNCIL ON MEDICAL EDUCATION AND HOSPITALS:
Dr. James S. McLester, Birmingham, Ala.
Dr. M. W. Ireland, Washington, D. C.

COUNCIL ON SCIENTIFIC ASSEMBLY:
Dr. Roger S. Morris, Cincinnati, Ohio

COUNCIL ON PHARMACY AND CHEMISTRY:
Dr. L. G. Rowntree, Rochester, Minn.
Dr. A. J. Carlson, Chicago, Ill.
Dr. Ernest E. Irons, Chicago, Ill.
Dr. W. McKim Marriott, St. Louis, Mo.
Dr. G. W. McCoy, Washington, D. C.

COUNCIL ON PHYSICAL THERAPY:
Dr. Ralph Pemberton, Philadelphia, Pa.
Dr. A. S. Warthin, Ann Arbor, Mich.
Dr. A. U. Desjardins, Rochester, Minn.

Dr. Rollin H. Stevens (Fellow), Detroit, was Chairman of the local Committee on Arrangements for the Detroit Session.

Dr. L. G. Rowntree (Fellow), Rochester, Minn., delivered a clinical lecture on "Arthritis" during the opening day. To the regular program for the Section on Practice of Medicine, the following Fellows contributed; Dr. Torald Sollman, Cleveland, Dr. Philip S. Hench, Rochester, Minn.; Dr. Ralph Pemberton, Philadelphia; Dr. L. M. Warfield, Milwaukee, "Hypothyroidism," Dr. T. L. Squier, Milwaukee, Dr. C. N. Hensel, St. Paul; Dr. James B. Herrick, Chicago, "The Clinical Signs of Heart Disease, with Particular Reference to Etiology," Dr. Sinclair Luton, St. Louis; Dr. Emanuel Libman, New York; Dr. M. W. Ireland, Washington; Dr. Charles H. Lawrence, Jr., Boston, "The Significance and Treatment of Menstrual Disorders in Adolescent Girls," Dr. C. J. Marinus, Detroit; Dr. O. W. Bethea, New Orleans, "The Treatment of Pneumonia," Dr. A. E. Greer, Houston; Dr. Ernest E. Irons, Chicago; Dr. Henry W. Woltman, Rochester, "Tumors Involving the Spinal Cord"; Dr. Stewart R. Roberts, Atlanta, "Agranulocytic Angina," Dr. J. E. Talley, Philadelphia; Dr. Herbert Z. Giffin, Rochester; Dr. James S. McLester, Birmingham, "Clinical Syndromes That Include Achlorhydria," Dr. O. H. Petty, Philadelphia; Dr. C. C. Bass, New Orleans, "The Treatment of Malaria, with Some Reference to Recently Promoted New Remedies," Dr. E. R. Whitmore, Washington; Dr. Joseph L. Miller, Chicago, the Frank Billings lecture on "The Present Status of Nonspecific Therapy"; Dr. George B. Eusterman, Rochester, Dr. H. L. Bockus, Philadelphia, Dr. G. G. Richards, Salt Lake City; Dr. Willard J. Stone, Pasadena, "Dietary Facts, Fads and Fancies"; Dr. Harlow Brooks, New York; Dr. F. G. Brigham, Boston, and Dr. S. S. Altshuler (Associate), Ann Arbor.

Many members of the American College of Physicians also contributed to other programs than that of the Section on Practice of Medicine. To the Section on Surgery, Dr. A. C. Ivy (Fellow), Chicago, gave a paper on "Physiologic Disturbances Incident to Jaundice." Dr. George E. Pfahler (Fellow), Philadelphia, discussed the paper on "The Use of Radium and High Voltage Roentgen Therapy in Conjunction with Radical Operation for Cancer of the Breast."

In the Section on Obstetrics, Gynecology and Abdominal Surgery, Dr. J. A. Myers (Fellow), Minneapolis, discussed the paper on "The Changes in the Mammary Glands of the Pregnant Albino Rat Deprived of Vitamin E." Dr. William Duncan Reid (Fellow), Boston, contributed a paper on "The Heart in Pregnancy." Dr. George R. Herrman (Fellow), New Orleans, with Dr. E. L. King, gave a paper on "Heart Disease and Pregnancy." Dr. A. J. Carlson (Fellow), Chicago, discussed the paper on "Pupillary Reactions as a Diagnostic Aid in Pregnancy."

In the Section on Ophthalmology, Dr. Gerald B. Webb (Fellow), Colorado Springs, discussed "The Eye in the Tuberculous Patient."

In the Section on Diseases of Children, Dr. Hugh S. Cumming (Fellow), Washing-

ton, reported on "The White House Conference on Child Health and Protection." Dr. C. C. McLean (Fellow), Birmingham, delivered a paper on "The Recurrent Incidence of Respiratory Infections of Childhood." Dr. J. A. Myers (Fellow), Minneapolis, discussed the paper on "The Significance of Advanced Tuberculous Infection in School Children."

In the Section on Pharmacology and Therapeutics, Dr. Roger I. Lee (Fellow), Boston, was a member of the Executive Committee. Dr. Soma Weiss (Fellow), Boston, delivered a paper on "The Treatment of Arterial Hypertension." Dr. Paul Dudley White (Fellow), Boston, gave a paper on "The Treatment of Edema by Mechanical Means," this paper being later discussed by Dr. Alpheus F. Jennings (Fellow), Detroit. Dr. Cyrus C. Sturgis (Fellow), Ann Arbor, with Dr. Raphael Issacs, gave a paper on "Treatment of Pernicious Anemia with Dried Stomach." Dr. Herbert Z. Griffin (Fellow), Rochester, with Dr. Charles H. Watkins, gave a paper on "Clinical Results in the Treatment of the Various Types of Secondary Anemia," this paper being discussed by Dr. A. B. Brower (Fellow), Dayton. Dr. Philip S. Hench (Fellow), Rochester, delivered a paper on "Unusual Reactions to Protein Therapy." Dr. Ernest E. Irons (Fellow), Chicago, discussed the paper by Doctors Cecil and Plummer on "Pneumococcus Type 1 Pneumonia, with Especial Reference to Serum Treatment." Dr. A. J. Carlson (Fellow), Chicago, discussed the paper by Doctors Van Dyke and Wallen-Lawrence on "The Growth Promoting Hormone of the Pituitary Body."

In the Section on Pathology and Physiology, Dr. A. H. Sanford (Fellow), Rochester, was Chairman, Dr. A. C. Ivy (Fellow), Chicago, Vice Chairman and Dr. J. J. Moore (Fellow), Chicago, Secretary. Dr. Aldred Scott Warthin (Master), Ann Arbor, gave a scientific exhibit on "The Pathology of Syphilis of the Heart and Aorta." Dr. Howard T. Karsner (Fellow), Cleveland, gave a lantern demonstration on "Pathology of Endocarditis." Dr. A. H. Sanford (Fellow), Rochester, delivered the Chairman address on "Role of the Clinical Pathologist." Dr. William Carpenter MacCarty (Fellow), Rochester, gave a lantern demonstration on "Principles of Prognosis in Cancer." Dr. John V. Barrow (Fellow), Los Angeles, gave a motion picture demonstration of "Characteristics and Pathology of Human Intestinal Protozoa." Dr. Sidney K. Simon (Fellow), New Orleans, Dr. Kenneth M. Lynch (Fellow), Charleston, and Dr. Frank Smithies (Master), Chicago, discussed Dr. Barrow's demonstration and paper. Dr. Samuel M. Feinberg (Fellow), Chicago, delivered a paper on "The Uses and Limitations of Skin Tests in Allergy," this paper being discussed by Dr. W. T. Vaughan (Fellow), Richmond, and Dr. W. W. Duke (Fellow), Kansas City. Dr. A. C. Ivy (Fellow), Chicago, discussed the lantern demonstration and paper by Doctors Mann and Bollman on "The Reaction of the Contents of the Gastro-Intestinal Tract." Dr. E. R. Whitmore (Fellow), Washington, was one of the discussants of the paper, "Chemical Studies of Malignant Conditions."

In the Section on Nervous and Mental Diseases, Dr. George W. Hall (Fellow), Chicago, was Chairman, Dr. Laurence Selling (Fellow), Portland, was Vice Chairman, Dr. Walter Freeman (Fellow), Washington, was Secretary, and Dr. Lewis J. Pollock (Fellow), Chicago, and Dr. George W. Hall (Fellow), Chicago, were members of the Executive Committee. Dr. W. H. Riley (Fellow), Battle Creek, discussed the paper on "Influence of Emotional Shock on the Gastro-Intestinal Tract in the Psychoneuroses." Dr. James L. McCartney (Fellow), Hartford, delivered an address on "Psychiatric Consultation Service Supplied by the State Department of Health." Dr. William C. Menninger (Fellow), Topeka, gave a paper on "Juvenile Dementia Paralytica: A Study of Forty Cases," which paper was discussed by Dr. Hans Reese (Fellow), Madison, Dr. Henry W. Woltman (Fellow), Rochester, and Dr. Paul A. O'Leary (Fellow), Rochester.

In the Section on Dermatology and Syphilology, Dr. Francis E. Senear (Fellow), Chicago, acted as Secretary. Dr. Paul A. O'Leary (Fellow), Rochester, was the discussant of the paper on "Syphilis of the Central Nervous System in Infants and Children." Dr. Maximilian A. Ramirez (Fellow), New York, with Dr. J. J. Eller, gave a paper on "Intradermal, Scratch, Indirect and Contact Tests in Dermatology: Comparative Study."

In the Section on Preventive and Industrial Medicine and Public Health, Dr. Francis M. Pottenger (Fellow), Monrovia, discussed the paper on "Racial Susceptibility to Tuberculosis." Dr. William Engelbach (Fellow), Santa Barbara, presented a paper on "Normal Weight and Measurements from Birth to the Age of Twenty."

In the Section on Orthopedic Surgery, Dr. Ralph Pemberton (Fellow), Philadelphia, gave a lantern demonstration of "Developments in the Problem of Arthritis."

In the Section on Gastro-Enterology and Proctology, Dr. Julius Friedenwald (Fellow), Baltimore, acted as Chairman, and Dr. A. F. R. Andresen (Fellow), Brooklyn, as Secretary. Dr. John A. Lichty (Fellow), Clifton Springs, gave a lantern demonstration on "The Present Care and Consideration of the Colon," which paper was discussed by Dr. Sara M. Jordan (Fellow), Boston, and Dr. John G. Mateer (Fellow), Detroit. Dr. Frank Smithies (Master), Chicago, gave a lantern demonstration, "From Medical Standpoint" in connection with the symposium on acute intestinal obstruction. Dr. Charles Eastmond (Fellow), Brooklyn, gave a lantern demonstration in the same symposium, "From Roentgenologic Standpoint." Dr. Anthony Bassler (Fellow), and Dr. J. Raymond Lutz (Fellow), both of New York, presented a paper on "Sprue: Diagnosis and Treatment." Dr. Samuel Weiss (Fellow), New York, gave a motion picture demonstration on "A New Gastroscope." Dr. George B. Eusterman (Fellow), Rochester, gave a lantern demonstration on "The Incidence and Diagnosis of Gastro-Intestinal Syphilis," in connection with the symposium on gastro-intestinal syphilis. Dr. Leon T. LeWald (Fellow), New York, gave a lantern demonstration in the same symposium on "Roentgen Diagnosis of Gastric Syphilis." Dr. Paul A. O'Leary (Fellow), Rochester, gave a clinical study on "Syphilis of the Liver." Dr. Julius Friedenwald (Fellow), Baltimore, gave the Chairman's address on "The Human Constitution in Its Relation to Gastro-Intestinal Diseases." Dr. Quinter Olen Gilbert (Fellow), Oakland, gave a lantern demonstration on "Some Evaluations of Gastro-Intestinal Motility." Dr. Martin E. Rehfuss (Fellow), Philadelphia, offered a lantern demonstration on "Acid Combining Values of Foods," which paper was discussed by Dr. A. C. Ivy (Fellow), Chicago, and Dr. Elmer L. Eggleston (Fellow), Battle Creek. Dr. A. J. Carlson (Fellow), Chicago, was a discussant of the paper on "Mechanisms of Gallbladder Contraction and Evacuation." Dr. H. L. Bockus (Fellow), Philadelphia, with Doctors Shay, Pessel (Fellow) and Willard, gave a lantern demonstration on "Diagnosis of Cholelithiasis Stressing the Relative Value of Nonsurgical Duodenal Drainage (Lyon Technique) and Cholecystography."

To the Section on Radiology, Dr. William B. Bowman (Fellow), Los Angeles, acted as Vice Chairman, Dr. George W. Grier (Fellow), Pittsburgh, as Secretary, and Dr. M. J. Hubeny (Fellow), Chicago, as a member of the Executive Committee. Dr. A. U. Desjardins (Fellow), Rochester, gave a paper on "Radiotherapy for Inflammatory Conditions," which paper was discussed by Dr. Rollin H. Stevens (Fellow), Detroit. Dr. Henry J. Ullman (Fellow), Santa Barbara, was the discussant of a paper on "Irradiation of Mammary Cancer, with Especial Reference to Measured Tissue Dosage." Dr. George E. Pfahler (Fellow), Philadelphia, with Dr. J. H. Vastine, gave a scientific exhibit on "Radium Therapy in Cancer of the Mouth with Especial Reference to the Newer Technic"; this paper was discussed by Dr. George W. Grier (Fellow), Pittsburgh. Dr. Albert Soiland (Fellow), Los Angeles, was one of the discussants of the lantern demonstration on "Indications and Limitations for Intensive Roentgen-Ray and Radium Treatment of Advanced Cancer." Dr. Soiland also gave an address on "Cancer Treatment." Dr. A. B. Moore (Fellow), Washington, and Dr. B. R. Kirklin (Fellow), Rochester, gave a paper on "Roentgenologic Diagnosis of Diaphragmatic Hernia." Dr. Sinclair Luton (Fellow), St. Louis, gave a lantern demonstration on the "Enlarged Heart: Its Detection and Significance," this paper being discussed by Dr. Leon T. LeWald (Fellow), New York. Dr. LeRoy Sante (Fellow), St. Louis, was the discussant of the paper on "The Roentgen Diagnosis of Small Pleural Effusions, with Observations of the Movement of Pleural Effusions."

OBITUARY

Doctor William Colby Rucker

In the untimely passing of Dr. W. C. Rucker (Fellow), May 23, 1930, the United States Public Health Service, the medical profession and the country at large loses an important figure in the field of medicine and sanitation. Dr. Rucker was comparatively a young man, at the peak of an active and progressive career, whose personality, scholarly attainments, executive and administrative ability gained for him an enviable reputation in varied fields. He was an indefatigable student, possessing a mind given to research and investigation. He was an author, a poet, a good clinician and possessed a subtle sense of humor which made his labors pleasurable and endeared him to all those with whom he came in contact. He contributed many articles and bulletins on public health subjects covering original work in epidemiology of the communicable diseases.

He was born on September 28, 1875, received his education at the University of North Dakota and the University of Minnesota where he was graduated in 1894. His medical degree he obtained from Rush Medical College at Chicago, Ill. He experienced a short period as commissioned officer in the Medical Corps of the United States Navy and in 1902 was given a commission in the United States Public Health Service. He received the degree of Doctor of Public Health at Stanford University, was a charter member of the Board of National Medical Examiners, and had the distinction of being both a Fellow of the American College of Surgeons and the American College of Physicians. As a sanitarian his reputation was international. He made many sanitary surveys in both Central and South America, played a leading and dominant part in the campaign against yellow fever in New Orleans in 1904 and was at that time a victim of the disease. In 1906, working with Dr. Rupert Blue in San Francisco, California, he conducted a splendid campaign against bubonic plague. While recuperating from this strenuous work, Dr. Rucker discovered, working with others, that squirrels could also transmit plague. In 1911 he was again a leading figure in the campaign against spotted fever at Victor, Montana. During the World War he was detailed to the American Expeditionary Forces where he did most creditable work and served until June, 1919. He was the first Chief Medical Advisor of the Bureau of War Risk Insurance at Washington, D. C., and helped to organize this department into an efficient organization. From 1920 to 1924, he was Chief Quarantine Officer of the Panama Canal. In 1925 he was detailed as Medical Officer In Charge of the United States Marine Hospital at New Orleans, La., at which hospital he served until his death.

Dr. Rucker developed into an extremely capable hospital head, was highly respected by the State and City hospital organizations and contributed heavily toward the organization of hospital superintendents. His pen was prolific in a wide range of non-technical subjects and his book entitled "Leadership," written largely for Serv-

ice officers, is accepted as a catechism for young officers entering the Service. He kept abreast with the medical profession of his time and rarely overlooked attending any type of medical gathering. He was 1st Vice President of the Association of Military Surgeons of the United States, a valued member of the editorial board of THE MODERN HOSPITAL and contributed consistently over a period of many years to this magazine.

His life was an inspiration to young officers in the United States Public Health Service and his *esprit de corps* advanced the Service which was so dear to him.

(Furnished by Randolph Lyons, M.D., F.A.C.P., Governor for Louisiana, through data furnished by William Y. Hollingsworth, M.D., and Waldemar R. Metz, M.D.)

Dr. Guy Lincoln Kiefer (Fellow), Lansing, Mich., died May 8, 1930, of angina pectoris; aged 63 years.

Dr. Kiefer received his A.B. and A.M. degrees from the University of Michigan, and later his M.D. degree from the same institution, 1891. In 1911, his Alma Mater conferred the honorary degree of Doctor of Public Health upon him.

During a long useful service, Dr. Kiefer served as County Physician of Wayne County; City Physician of Detroit; Professor of Preventive Medicine and Public Health at the Detroit College of Medicine and Surgery, Chairman of the Section on Preventive Medicine and Public Health of the American Medical Association and Vice Chairman of the Section on Public Health Administration of the American Public Health Association.

He was an Ex-President of the Michigan State Medical Society and of the Wayne County Medical Society, a Fellow of the American Medical Association and had been a Fellow of the American College of Physicians since 1917.

Dr. William J. Kay (Fellow), Lapeer, Mich., died April 16, 1930, of streptococcic sore throat; aged, 63 years.

Dr. Kay was born at Belmore, Ontario, and received his preliminary education at the Harriston Collegiate Institute. He graduated in medicine from the Detroit College of Medicine and Surgery in 1897. For several years, he has been Consulting Internist and Medical Superintendent of the Michigan Home and Training School. He was a member of Lapeer County Medical Society, an Ex-President of the Michigan State Medical Society, a Fellow of the American Medical Association and had been a Fellow of the American College of Physicians since 1919.

Dr. Robert Pollock (Fellow), San Diego, Calif., died, June 2, 1930, aged 64 years.

Dr. Pollock was a graduate of the Western Reserve University School of Medicine, 1892, and interned at the Cleveland City Hospital in 1892-93. He went to San Diego nineteen years ago. His health, which had not been good, returned and he again took up active practice and played an important part in bringing medicine to its high position in San Diego. He was

active in his local medical societies, had acted as a delegate to state and national organizations and was an Ex-President and member of the Board of Governors of the Southern California Medical Association. His untiring effort lives as a monument in the San Diego Medical Library for which he did so much. His good counsel and earnest efforts for better medicine will be missed. The medical profession in California and the citizens of his community will miss a loyal citizen and a good physician.

(Furnished by: Egerton Crispin, M.D., F.A.C.P., Governor for Southern California.

Dr. Antonio D. Young.

On June 3, 1930, the State of Oklahoma suffered a distinct loss through the death of one of its most useful citizens. Removed from the turmoil of traffic, on a shaded street, in the upper chamber of his quiet home, Dr. Antonio D. Young died, as he had lived, with commendable courage and composure.

Here was a man who loved his fellow men and though he lived well within the plane of their comprehension he was set apart as the kindly physician. While highly trained and peculiarly skilled in his chosen specialty he never lost sight of the fact that sick people need a physician with a practical knowledge of the human body, who is willing to sit at the bedside and bring to bear a sympathetic, intelligent application of this knowledge to the patient's individual needs.

Dr. Young possessed a radiant personality; to look into his eyes; to receive his genial smile; to hear his voice and shake his hand was enough to send one joyfully on his daily round of duties.

Though by no means a recluse he avoided fame, always insisting upon remaining on his accustomed level, where with simplicity and gentleness he enveloped all with his tolerant insight and sympathetic understanding, saturating his environment with the most delicate wit and humor.

In closing this memorial we can think of nothing more appropriate than the following from Maurice Maeterlinck: "Our dead are greater and more truly alive than we are; when we forget them it is our whole future that we lose sight of; and when we fail in respect to them it is our immortal soul that we are trampling under our feet."

Dr. Young was born in Jerseyville, Ill., December 11, 1873, and graduated from the Jerseyville High School in 1892. He graduated at Barnes Medical School, St. Louis, Mo., in 1895, and practiced medicine at Downs, Ill., until he came to Oklahoma City, February, 1901. He was married to Elberta Meyer, December 1, 1897.

Dr. Young was secretary of the faculty of the Medical Department of Epworth University which later became the Medical Department of the Oklahoma State University. He was professor of Neurology in the latter school from the date of its organization.

Dr. Young served in the World War from March 1918 to 1919.

He was a Fellow of the American College of Physicians, a member of the American Medical Association, the Southern Medical Association, the Oklahoma State and County Associations

and the Oklahoma City Academy of Medicine. He was a charter member of the latter and helped write its constitution. He was also a member of the Men's Dinner Club and was closely indentified with various social and outdoor clubs.

(Furnished by: Lea A. Riely, M.D., F.A.C.P., Governor for Oklahoma.)

Dr. Frank Canfield Hollister (Associate), New York, N.Y., died suddenly November 30, 1929, aged 64.

He was a graduate of the Bellevue Hospital Medical College, Class of 1890.

Dr. J. Edward Harbinson (Fellow), Woodland, Calif., died during April, 1930. Dr. Harbinson was born, April 28, 1895, in Yolo County (California), and was educated in the Sacramento Public Schools. He received his Degree of Bachelor of Science in the University of California in 1917, and his Degree of Doctor of Medicine from the same institution in 1922. He was Interne and Assistant Resident Physician at the University of California Hospital during 1922 and 1923, and then became Physician in Chief at the Woodland Clinic Hospital, which appointment he held until the time of his death.

Dr. Harbinson contributed numerous scientific articles to medical literature; these articles dealing chiefly with the use of Amiodoxyl Benzoate in the treatment of Arthritis and Undulant Fever.

He was a member and Ex-President of the Yolo-Colusa County Medical Association, a Fellow of the American Medical Association, a member of the California State Tuberculosis Association, and was elected a Fellow of the American College of Physicians on October 27, 1929.

Dr. Joseph McIntyre Patton (Fellow), Chicago, Ill., died, April 16, 1930, of chronic myocarditis, arthritis and arteriosclerosis; aged, 69.

Dr. Patton was born at Ralston, Pennsylvania, received his preliminary education at Hasbrouck's Institute of Jersey City and graduated from the Medical Department of the University of the City of New York in 1882. He was Professor Emeritus of Clinical Medicine at the University of Illinois, College of Medicine, and in earlier years was Professor of Physical Diagnosis in the College of Physicians and Surgeons of Chicago. Dr. Patton was the author of a long list of publications, which appeared in the leading medical journals of the country. He was author, also, of two books, "Clinical Lectures on Heart, Lungs and Pleura" and "Anesthesia and Anesthetics." He was a Fellow of the American Medical Association, a member of the Illinois State Medical Society, a former President of the Chicago Medical Society, and had been a Fellow of the American College of Physicians since 1920.